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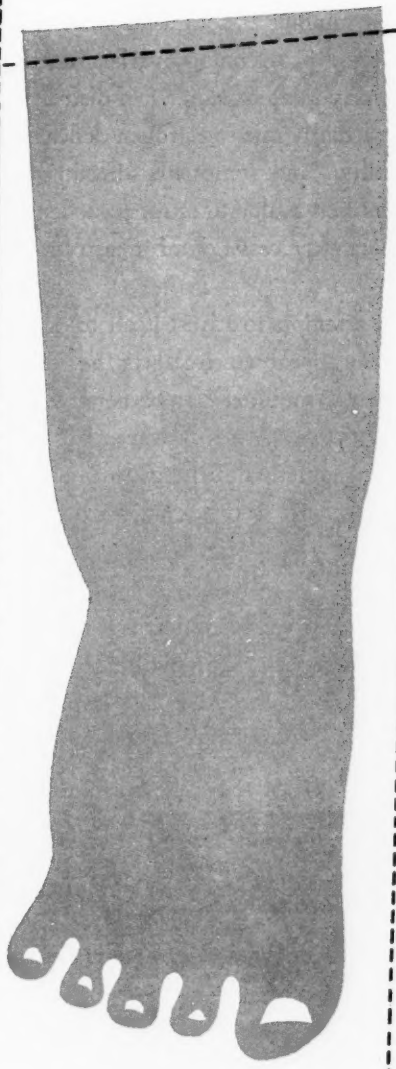

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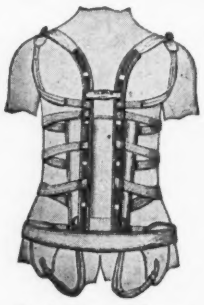

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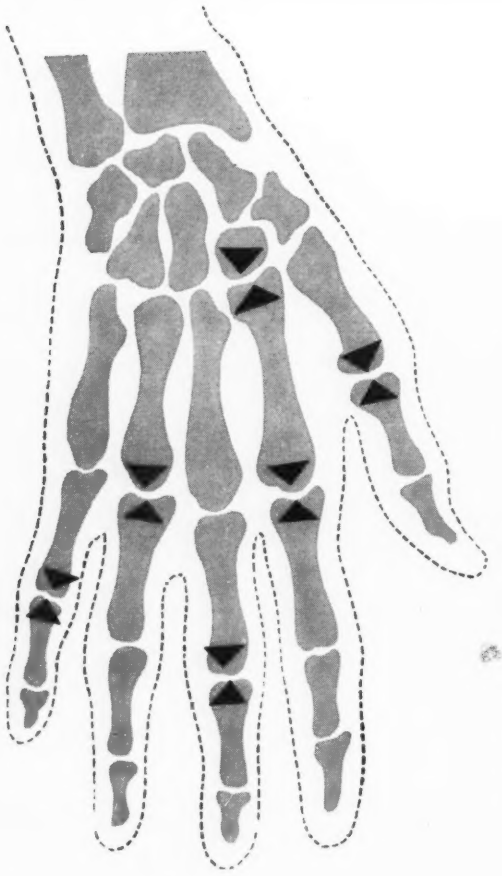

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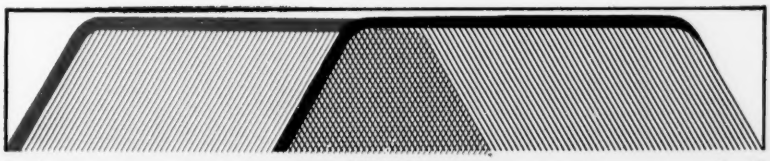

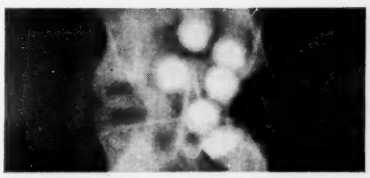

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Complete Heart Block with Morgagni-Adams-Stokes Syncope Treated with Large Doses of Epinephrine: A Case Report

William E. Larsen, M.D.
Kansas City, Kansas

The purpose of this paper is to illustrate the dosage level of epinephrine sometimes needed in the therapy of Adams-Stokes syncope of ventricular standstill origin complicating complete heart block.

CASE REPORT

A 48-year-old white man entered the hospital because of frequent "black-out" spells. He was perfectly well until five days prior to admission, at which time he noted the onset of a sore throat, malaise, and a low anterior pain traversing the chest. These symptoms followed an exposure to inclement weather. The following day the patient had one transient episode of fainting while lying in bed. His condition was considered to be due to "viral flu," and he was given antibiotics. From that time until his admission to the hospital, he had fever, profuse diaphoresis, anorexia, lethargy, and a low anterior chest pain which, on the day prior to admission to the hospital, radiated into his left shoulder. On the morning of admission he had three fainting spells of short duration, and for five hours preceding admission he had frequent tonic and clonic convulsive episodes associated with periods of unconsciousness.

The past history was non-contributory to his present illness, except that when he was in the military service in 1940 he was told that he had a slow heart rate. The patient's family and social history were not unusual.

The physical examination upon admission revealed an acutely ill, white man of 48 years of age. His temperature was 100.2 degrees F., his pulse rate was 56 per minute, and his respiratory rate was 16 per minute. Initial observation revealed the patient to be well developed and well nourished, and lying in bed with frequent episodes of syncope accompanied by tonic and clonic convulsions. He was extremely apprehensive. The convulsive episodes occurred at about five- to ten-minute intervals and were of 45 to 60 seconds duration. The lung fields were clear to percussion, and there were fine moist crackling rales heard at both bases posteriorly. The heart rate was 56 per minute and regular. The first mitral tone varied in intensity from beat to beat, and no murmurs were heard.[5] The pulse and heart sounds became imperceptible about five seconds prior to the episodes of syncope. The remainder of the examination was not unusual.

Laboratory examination showed a white blood count of 19,800 per cu. mm. of which 80 per cent were neutrophiles, 12 per cent were monocytes and 7 per cent were lymphocytes. The red blood count was 4.26 million per cu. mm. with 12 grams of hemoglobin. The erythrocyte sedimentation rate was

This paper, submitted for the March (University of Kansas School of Medicine) issue, was omitted then because of lack of space. Dr. Larsen is from the Departments of Medicine, the Veterans Administration Hospital, Kansas City, Missouri, and the University of Kansas Medical Center, Kansas City, Kansas.

42 mm. in one hour. The blood serology was negative, and the urinalysis was normal. A portable chest x-ray revealed an increased density in the lower left chest. A repeat chest x-ray on the seventh hospital day showed normal lung fields. An electrocardiogram taken shortly after admission (Figures 1 and 2)

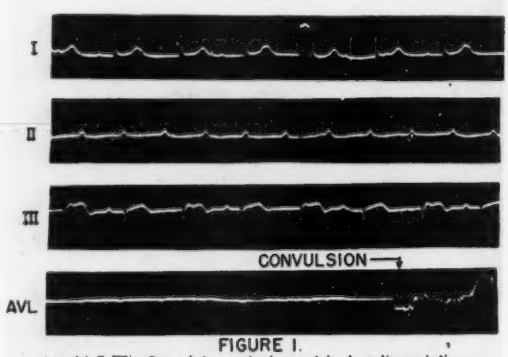

FIGURE 1.

Lead I & III Complete auriculo-ventricular dissociation.
Lead II Auricular complexes with ventricular standstill.
Lead AVL Auricular complexes with ventricular standstill leading to a convulsion.

Electrocardiogram taken on the first hospital day showing complete auriculo-ventricular dissociation and ventricular standstill.

showed a slow, regular rhythm with a complete disassociation between the auricular and ventricular complexes indicating a third degree heart block. In Figure 1 there are only auricular complexes seen in lead II and aVL. There is an elevation of the RS-T segment in leads II, III and aVF with a depression of the RS-T segment in lead V-2. The interpretation is a third degree heart block with periods of ventricular standstill and evidence of an early posterior myocardial infarction.

The patient was placed on therapy of epinephrine, 1:1000 dilution, 0.3 cc. subcutaneously every hour. Because of continued episodes of ventricular standstill and convulsions, it was soon evident that more frequent and larger doses of epinephrine would be necessary. Consequently, the dose was increased to 0.4 cc. subcutaneously every 30 minutes, and ephedrine sulfate 45 mg. was given orally every four hours. Only at this dosage level was the patient free of syncopal attacks.

By the third hospital day, the patient had been free of syncope for 24 hours. The dosage of epinephrine was gradually reduced and was discontinued on the fourth hospital day. With this amount of epinephrine, which totaled 20 cc. of 1:1000 dilution in one 24-hour period and 47 cc. over a period of three days, the patient did not become excited and agitated but had a relaxed feeling as well as relief from apprehension.

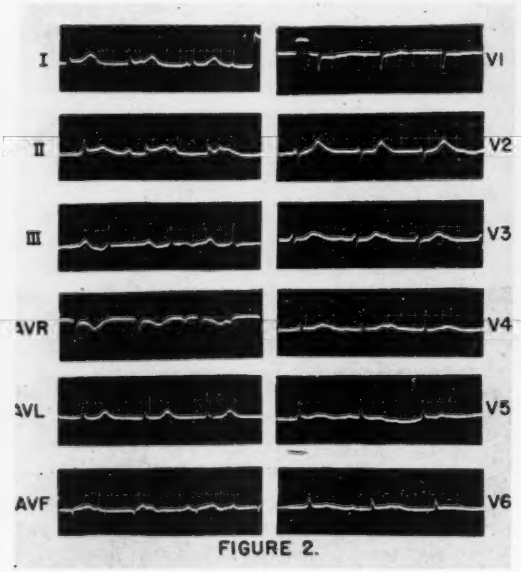

FIGURE 2.

Electrocardiogram taken on the first hospital day showing a complete auriculo-ventricular dissociation.

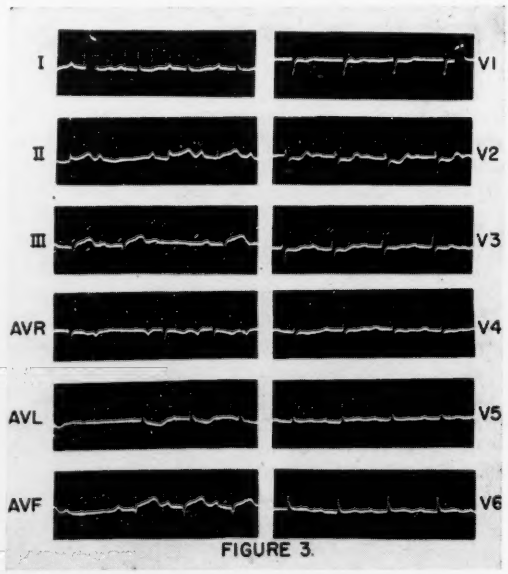

FIGURE 3.

Electrocardiogram taken on the third hospital day showing a second degree heart block with a Wenckebach phenomenon.

Serial electrocardiograms (Figures 3, 4, and 5) revealed gradual recovery from the complete heart block through second and first degree heart block to a normal sinus rhythm. In Figure 3, it will be noted that a Wenckebach phenomenon is present. The persistence of RS-T changes in lead II, III and aVF confirmed the original impression of a posterior myocardial infarction.

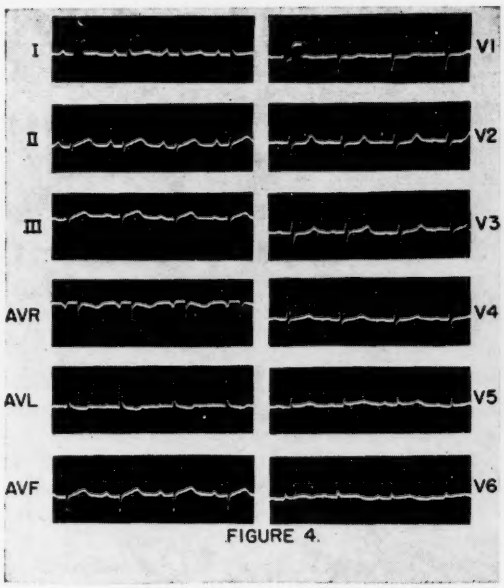

FIGURE 4.

Electrocardiogram taken on the sixth hospital day showing a first degree heart block and evidence of a posterior myocardial infarction.

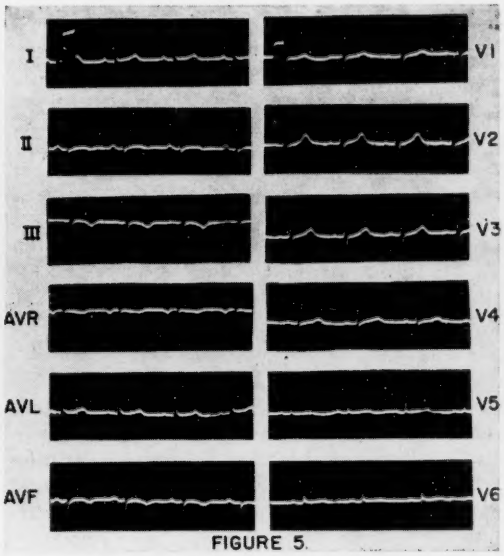

FIGURE 5.

Electrocardiogram taken on the 12th hospital day showing normal A-V conduction and a posterior myocardial infarction.

The progress of the patient was satisfactory, and he was discharged on his 21st hospital day to continue his convalescence at home. A follow-up examination eight months after his acute illness revealed an apparently healthy man with electrocardiographic evidence of myocardial insufficiency and an old posterior myocardial infarction.

DISCUSSION

The rhythm of the ventricular muscle is normally influenced in three manners: (1) via the conductive pathways within the Purkinje system of the heart; (2) by sympathetic fibers via the superior, middle, and inferior cardiac nerves and direct fibers from the upper four and five thoracic ganglia, and (3) by hormonal influence of circulating epinephrine. When the normal pathways are interrupted, an inherent focus in the septal myocardium (A-V node) takes over the function of a rhythmical pacemaker. All myocardial tissue has this inherent potentiality of rhythmicity.

The stimulation of the sympathetic nerves to the heart or an increase in circulating epinephrine to the heart results in (1) an accelerated and more vigorous beat, (2) a shortening of the refractory period as well as shortening of the mechanical and electrical systole, and (3) an increase in the automaticity of all pacemaking tissue. It is this last function of epinephrine which is important in the therapy of ventricular standstill of complete heart block.

When one is confronted with a problem of Morgagni-Adams-Stokes syncope, it is of extreme urgency to determine the cause of the attack associated with an imperceptible pulse because the therapy of ventricular standstill could be disastrous if applied to a case of ventricular tachycardia or fibrillation. The only means of differentiation is the electrocardiographic record during an attack of syncope.

The quantity of epinephrine which was necessary in this case to maintain a regular cardiac rhythm free of asystoles was far in excess of recommended dosages. However, in a case such as this one, to obtain the desired therapeutic effect, the usual dose must be increased several fold. The assay of epinephrine used was verified to be of full potency.[13] During the administration of epinephrine, none of the other effects such as tremor, nervousness, or anxiety was noted.

SUMMARY

A case of Morgagni-Adams-Stokes syncope complicating complete heart block secondary to a myocardial infarction is presented, and the therapy using large doses of epinephrine is reviewed. In one 24-hour period, 20 cc. of 1:1000 epinephrine was needed, and over a period of three days 47 cc. was necessary.

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The Art of Medicine and the Cancer Patient

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When a patient is presented with the diagnosis of cancer for which specific measures are not immediately initiated nor advisable, and when this patient returns to his home, the local doctor is confronted with a real problem in philosophy and medical management.

Having been presented with such patients, the writer has evolved several bits of philosophy which are adamant. First, and most prominent, it is felt that a definite prediction as to the time of the patient's death is not within the province of any doctor. The outlining of "time" either to the patient or to the patient's relatives does not seem to be medically correct in that the endurance of that particular individual is an unknown; in that the course of any cancerous cell is not predictable even with histological support; and in that the variables of cancerous cells *in situ* are too numerous to permit accuracy of prediction.

In essence, it is felt that the greatest injustice that a surgeon, a radiologist, or any doctor can do to the patient, to the family, to himself, or to the family doctor, is to make a definite pronouncement regarding the probable time of dying. The patient returns home with a psychotic noose around his throat—the rope being tightly secured—so the family doctor has little hope of overcoming the fear of the predicted time. Any efforts toward encouragement are thwarted by previous and more authoritatively received words.

Every doctor can recall among his patients some one patient who spent several months "dying," only to go on and live for years; in fact, the patient finally and actually dies of "other causes." A specific example—a woman underwent surgery in Topeka in early 1947. She was informed directly and indirectly that six months was the most time possible for a patient with her type of cancer to survive. Therapeutic x-ray was administered, some supportive medications were given, and the patient was requiring paracenteses for abdominal ascites when first seen—two years later—in November 1949.

The patient was seen at her daughter's home in Topeka as a courtesy. The first impression—even in consideration of the ascites—was that the patient was more discouraged than she was terminal in her appearance. In addition, a herpetic lesion over the left eye made life's noises the more annoying to her. Words of encouragement were given. It was implied that she could yet spend Thanksgiving and even Christmas in her own home, and a detailed written program of living, eating, and medication was outlined with a definiteness that re-enforced the idea that it was worth the effort and that "life was expected to continue."

The patient spent that Christmas (1949) in her own home. The paracenteses were less frequently necessary. In fact, the patient and her husband were able to revisit their childhood communities during the next summer. They were able to spend another Christmas (1950) fairly pleasantly and in their own home. The woman quite suddenly became ill in January 1951 and died within a 48-hour period.

It is true that some 120 therapeutic x-ray treatments were given after surgery and before November 1949. It is likewise true that a total of 17 paracenteses were required from November 1949 until January 1951. Conversely, it must be stated that the woman spent 6 months dying, another 12 months wondering why she hadn't, and another 24 months living an apparently well adjusted life—a pleasant woman who resumed her practices of sending candies and treats to friends, neighbors, and children.

Parenthetically it is recorded that the daughter's husband, in whose home the patient spent her first 18 months of illness, packed up and disappeared without leaving any word as to his destination. To my knowledge, he has never returned to his wife and children. The entire story might have read differently if "hope" had been expressed after surgery rather than "time" having been impressed.

Written at the invitation of the Committee on Control of Cancer, Kansas Medical Society.

This paper proposes to outline a practical philosophy toward the management of such problems. It is hoped that patients and relatives have not been too thoroughly "doomed" to be unredeemable.

The philosophy of Sir William Osler in his lecture, "A Way of Life," is appropriate—"to live in day-tight compartments." Or, to quote Carlyle, "Our main business is not to see what lies dimly at a distance, but to do what lies clearly at hand." The future is today! This philosophy can be skillfully injected even into a cancer-minded patient.

The patient's cupboard is too often too full of pills for morning, afternoon, and bedtime; the room is too empty of the elements of encouragement to continue to live for today and each day. A positive attitude toward what to do, what to eat, and where to go is beneficial to the patient, his family, and even to the doctor. The end of any life being unpredictable, energies are better expended on encouragements than upon the "hypos" of discouragement.

Specific areas of preachment are believed to be practical and acceptable to the patient and to the relatives. Thus, it is proposed that a positive preachment of an adequate diet be made; that the seven basic foods be presented; that the value and necessity of regular meal times be stressed; that the food be well prepared, attractively served, and actually eaten.

One is reminded of the story of the political prisoner who dressed formally for each meal, ate of the food served him, and outlasted by some 20 years all of the others who were originally imprisoned with him. It is felt that the time and attention given by the doctor to stressing the necessity of adequate meals, actually eaten, will be repaid by a patient who becomes more co-operative and stays happier longer.

Secondly, there is considerable medical writing concerning the probability of cancer being a "deficiency" disorder. Similarly, there are many articles in the lay press which impart these same thoughts. The pens of authors who are trying to publish medical papers in the lay press and the pens of the various faddist groups continuously promote this thinking. In either case, the positive approach to an adequate diet usually meets with the family's approval and is physically and psychologically desirable for the patient.

I recall a family having several lawyer sons who commented to the doctor, "Now that the scientific measures have been outlined, just what do we feed the man?" It may be a correct accusation—that we are overzealous in our medical management and are unwittingly negligent in simply feeding the patient.

In medical writing, an adequate diet can be advocated by nutritionists as being proper for the so-called healthy adult. Much work is being done on the normal requirements of the geriatric patient. It would seem proper to feed such patients at least a normal diet.

Within the field of patients having Hodgkin's disease, it is reported that Bacillus acidophilus, oral penicillin, and oral sulfathiazole alter the intestinal tract to a situation in which the patient feels better and the usual anemia is more easily controlled. It is not contended that the course of the cancer is altered, but it is noted that the immediate well-being of the patient is improved. Quite often the patient becomes intrigued with dietary possibilities and will read with alertness and real interest every available article. He continues to live.

Other research would hint that the various sources of vitamin B should be abundantly supplied. The drinking of an abundant amount of water by count and not simply by thirst often freshens the patient remarkably.

In short, regardless of the supposed scientific "value-less-ness" of these suggestions, attention to an adequate meal plan, attention to possible favorable products, and attention to rules which should apply to general health all make sense to the patient. Something is being done by him and for him. Ideas have replaced the "emptiness" of awaiting a predicted fate. Foods, vitamins, tonics, and amphetamine buoyants can be used much longer and with much more acceptance than are the usual depressant bromides, barbiturates, and narcotics.

Again, encouragement to live today, and to do today as much of as many things as you can is definitely a form of treatment. It can be commented that it is never safe to be alive; nevertheless, the patient must be encouraged to live. He must be encouraged to continue his vocation and his hobbies. The financial ship should be made more secure and more definite, but it should not be abandoned. There is a great difference between having legal matters in order and the drawing of a "finis" line on the entire endeavor. Similarly, it is better to plant the bush and never see the rose than it is to quit planting.

A by-product of this writing can be the admonishment of the medical doctor to preach encouragement, to predict the possible continuation of living, and to avoid the "emptiness" of looking only to life's termination. A program of "nothingness" is felt by the patient and the family to the extent that they are actually wedged into the care of faddists and cultists who do promise and who do see that something is actively being done.

We have failed as physicians when we forsake a patient who is ill with cancer and when we, by negativism, cause him to turn to other groups for care. So-called modern medicine, together with personalized encouragement, should be superior to the too-often administered colonic irrigations.

Throughout this writing there are no new suggestions as to specific therapy. It was assumed that the diagnosis implied failure of our scentific endeavors. Contrariwise, there is the definite thought that we should be wary of suggesting a time of life's termination to any and every such patient, but rather that we should adopt and positively present an active program to these patients, that a positive outlook on each day of life is desirable and possible, that enforcement of an adequate diet is favorably received by the patient, and that a period of relatively satisfactory living can be inserted between the time of diagnosis and the time that final supportive care becomes necessary.

A Case of Actinomycosis of Ovary

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It has been aptly said that, in the practice of medicine, it would be better not to diagnose the rare and unusual until the common well known ailments can be ruled out—also that the only excuse for publishing a scientific article is to add to, or simplify, preexisting knowledge. However, the rare and unusual promptly finds its place in the literature when it does occur, and the following presentation of a case of ovarian actinomycosis is along this line.

Actinomycosis is a disease caused by a fungus which grossly appears in the form of "sulfur granules" lying in small pockets of pus; microscopically it consists of club shaped filaments arranged radially like the sun's rays, hence the name "ray fungus."

Grossly, its lesions are a suppurative granulomatous process more commonly seen in cattle, located at the angle of the jaw and popularly known as "lumpy jaw." The earliest report of the disease was in 1845. Transmission from cattle to human beings has not been proved. Most of the human cases occur in persons who have had no contact with diseased animals.

It has been suggested that minor traumatism, as from fragments of grain imbedded in the soft tissue of the mouth, may be a factor in the infection.

In the earlier reports it was considered that the source of infection might be *exogenous* or *endogenous,* in the former being due to chewing grasses, grains, or cornstalks. In the latter it is believed that the fungus is a "normal inhabitant" of the alimentary tract, where, aided by minor trauma and perhaps by secondary invaders in the form of cocci or bacilli, it may attack the human body. Since the ray fungus is an obligate anaerobe, the exogenous theory of infection is untenable. However, another species of the Actinomyces group called "Nocardia" is an aerobe and causes lesions similar to Actinomyces bovis. Its lesions are called "nocardiosis," said to begin most often in the lungs but spreading to other parts of the body.

From this, one might get the reaction that "a rose by any other name is just as sweet," or postulate a theory of one's own that the ray fungus is a "Dr. Jekyll and Mr. Hyde" type of organism living first as a facultative anaerobe, an innocuous exogenous existence under an assumed name (Nocardia), and becoming dangerous only as an obligate anaerobic organism (Actinomyces bovis) under favorable conditions as previously noted.

From its primary location in one part of the body, it is believed that the organism may be spread to other organs by contact or through the blood stream. Some believe that the primary source of pelvic lesions may be the vagina (use of pessary or abortion followed by infection). Some cases have even been believed to be primary in the pelvis. The pelvic organs, when invaded, consist of ovaries and tubes with occasional involvement of parametrium. When only one ovary is affected, the right is reported as being most often involved. (Previous operation such as appendectomy followed by adhesions is found in many cases.) Involvement of pelvic organs occurs chiefly in women before the menopause. Characteristic laboratory findings: r.b.c. normal, mild hypochromic anemia, elevated w.b.c., sedimentation rate increased, urine apt to contain pus. Diagnosis (in suspected cases) is confirmed by microscopic examination, cultures, and microscopic examination of tissues.

Mrs. O. H., housewife, age 37, consulted her physician July 25, 1953, complaining of pain in epigastric region, accompanied by chills, fever, and abdominal distention. She had had morning sickness for three weeks and thought she might be pregnant. (Frog and rabbit tests produced prompt death of the animals.) She had lost 30 pounds in weight, unaccountably.

Physical examination revealed abdominal distention and a mass in the pelvis which was sore and tender. The impression was pelvic inflammatory disease, probably salpingitis.

Vaginal examination August 1, 1953: Cervix long,

not fixed and open, no mass nor tenderness on right side, left parametrium tender, and suggestion of mass left of uterus.

Operation (10 days after onset): Mass involving both tubes, ovaries, and parametrium, filled with one cup of white odorless pus. The patient was given lots of antibiotics and bled profusely during the operation, requiring several blood transfusions.

She left the hospital on the 13th postoperative day and had some elevation of temperature one day afterward. She was still draining on August 21, 1953, but had no elevation of temperature, and antibiotics were stopped.

Summary of pathologist's report (specimens hardened in formalin): Specimens consist of two ovaries and tubes. The larger (left) ovary is 4 x 4 x 2½ cm. containing a previously opened 2 cm. cavity at one pole suggestive of abscess. Much of the adjacent ovarian tissue is pale yellowish-white and on gross section shows numerous tiny orange-colored granules imbedded in its substance, but no grossly visible pus. An occasional follicle cyst is seen in the cortex. The corresponding tube is 1¼ cm. thick along its distal third. The ostium is patent.

The right ovary is slightly smaller with congested surface and contains multiple follicle cysts (up to 4 mm.). The corresponding tube is 1 cm. thick with patent ostium (Figure 1).

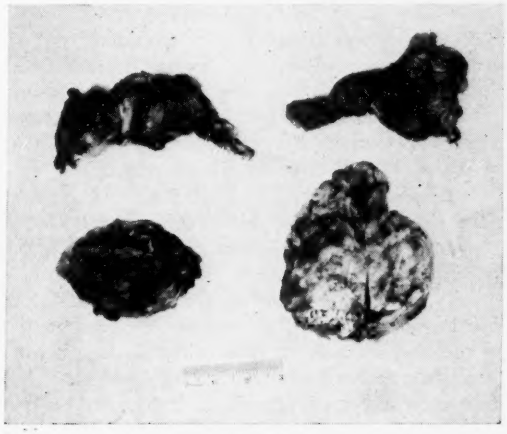

Figure 1

Microscopic sections of the left ovary show, in addition to wall of abscess noted grossly, numerous ray fungi surrounded by pus cells (Figure 2).

The opposite ovary shows mild active inflammation. No fungi are found in this ovary.

Sections of the tubes show swollen mucosal folds thickly infiltrated by neutrophiles and plasma cells. The lumens contain purulent exudate. No fungi are seen in the sections examined.

Diagnosis: actinomycosis of left ovary; active inflammation (mild) of right cystic ovary; bilateral pyosalpinx.

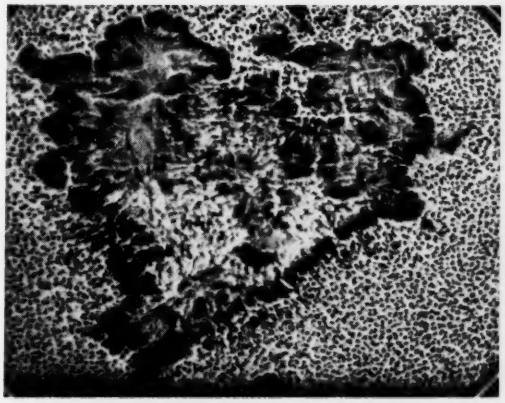

Figure 2

The patient was later admitted to the Mayo Clinic, and she was again operated upon. Details are unknown at present. She was dismissed about the last of October and returned home, reported to still have a draining sinus from the pelvis through the skin. She was advised to return to the Mayo Clinic after a period of time for a check-up.

Comment: A rare case of human actinomycosis involving only the left ovary, with bilateral pyosalpinx and mild active inflammation of the opposite ovary, is presented. The case should be of interest to surgeons and gynecologists. The disease is particularly to be suspected in cases with a history similar to that presented, when the condition does not yield readily to antibiotics and is followed by persistent draining sinuses following operation. The ray fungus is hard to culture, rendering tests for sensitivity to various antibiotics difficult. Sulfadiazine and penicillin seem to be the ones most used.

Prior to the advent of antibiotics and chemotherapy, KI was the chief therapeutic agent. It is reported that with antibiotics the prognosis is better, but actinomycosis is considered a serious condition with high mortality.

According to Herbut, slightly over 100 cases of actinomycosis of tubes and ovaries had been reported by 1949. He states that the intestinal tract is probably the primary site of infection.

Numerous details regarding acid fast characteristics of the fungi, characteristic clinical laboratory findings including increased sedimentation rate, etc., have been omitted in this presentation, but they may be found in the previous literature on this subject.

Much appreciation is due Dr. W. W. Weltmer of Beloit, Kansas, and Dr. C. A. Hellwig of the Hertzler Clinic for their help in preparing this article.

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Guillain-Barre Syndrome: Report of Two Cases Treated with Vitamin B Complex, Cortisone, and ACTH for Relief of Pain

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A number of excellent reviews of the literature on Guillain-Barré syndrome have been published by Hand and Rudoy,[5] MacNeal and Bland,[8] and Stillman and Ganong[10] within the last few years. Pathological reports by DiFiore[2] and Lowenberg and Foster[7] quite adequately explain its protean clinical manifestations on the basis of distribution of the pathological lesions.

Although this condition shows many of the characteristics of a specific disease entity, we must consider it a syndrome until more definite causative factors can be determined.

Prior to Guillain, Barré, and Strohl's[4] first paper in 1916, many cases had been reported under such headings as infectious neuritis, infectious polyneuritis, radiculitis, acute ascending myelitis, Landry's paralysis, myeloradiculitis, neuritis, neuronitis, and many other terms. It remained for the above authors to call attention to the characteristically high spinal fluid protein with a normal or at least relatively low cell count.

The onset of this condition is usually fairly acute and frequently follows an upper respiratory infection. However, in this clinic we have had one case that occurred shortly after a severe gastric hemorrhage and another that terminated fatally following a gallbladder operation. Most patients give the history of a fairly acute onset beginning with decrease in motor function and sensory loss accompanied by paresthesia. Muscle pain may be present in varying amounts. In one of our patients, constant severe back pain and numbness of the fingers were present two weeks before weakness was observed.

DiFiore reported a series of cases which well demonstrates the variable clinical picture. In his series he reports one patient with a monoplegic involvement, another who was paraplegic, an ascending type of onset, and a generalized type (with or without facial diplegia). The onset is usually accompanied by a low grade fever and very little change in the blood count, and it apparently runs a self-limited course which is, of course, suggestive of an infectious disease.

When death occurs, it is usually due to respiratory paralysis. Consequently, one should always have a respirator readily accessible to the patient. Once the patient gets over the acute phase, convalescence is usually continuous although frequently prolonged, due to the length of time required for regeneration of the long nerve fibers. Residual paralysis or permanent damage has been reported in some cases, but frequently recovery is complete.

The two cases we are reporting were of the generalized type with sixth nerve weakness and severe pain. Both cases have made a complete recovery with no residual changes. However, they did have more pain than is usually present. In one patient the pain was limited to the back, while the other patient complained not only of severe back pain, which was continuous, but of a severe, sharp, stabbing pain in all extremities. In fact, it was this patient's severe pain that led us to consider the use of ACTH.

As stated above, the cause of Guillain-Barré syndrome is not known. Gavin[3] and Kennedy[6] have sug-

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gested that it could be an acute infectious process. In that case, ACTH or cortisone would certainly be contraindicated; on the other hand, Reitmen and Rothschild[9] believe that the condition is the result of an allergic phenomenon, and of course in such a case it would be the treatment of choice. A review of the literature revealed one case successfully treated by Stillman and Ganong.

CASE 1

Mr. W. B., a 43-year-old white married farmer, was referred to the Halstead Hospital by Doctors D. A. Hardman and L. W. Bauer of Smith Center, Kansas, on February 11, 1952. He came with the complaints of numbness in the fingers and toes, fatigue, irritability and pains in the back and all over the body, with onset two to three weeks previous.

Present Illness: The patient had not slept well the past two months, but his appetite had been good. At Christmas time, six weeks before admission, an incident occurred in this man's personal life which he found upsetting and frustrating at that time and about which he worried. However, at the onset of this condition he felt that he had come to accept the matter and was taking it in his stride.

Past History: The patient had no emotional upsets or serious illnesses in the past. He had sciatica a few years before which was quite painful, but he had had no trouble for several years. The family history was negative.

At the time of the physical examination, the patient was mentally dull, probably due to recent medication; however, he was constantly twisting and turning in bed, trying to find a more comfortable position. He complained of weakness and pain in the legs a week prior to admission and described this as a severe, constant type of pain. He also complained of sudden, sharp, shooting pains in all his extremities but principally in the right leg. Examination of the patient on admission revealed a well developed, well nourished, rather muscular man. The neck was stiff. The straight legs could not be elevated more than 35 degrees. The patient exhibited a great deal of soreness in the muscles of the calves of both legs and in the right thigh and back. Pupils were equal, regular and reacted to light and accommodation; extraocular movements were normal. There was no complete paralysis of any muscles; the grips of both hands were weak; he showed considerable weakness in his legs, could barely lift the heel off the bed; biceps, Achilles, and knee jerks were markedly decreased; there was a suggestive Babinski's sign on the right; abdominal and cremasteric reflexes were absent. Temperature was 98°, pulse 70, blood pressure 130/80; heart, lungs, and abdomen were negative.

Laboratory Findings: Urine negative. Sedimentation rate 20 mm. in the hour. Hemoglobin 84 per cent, rbc. 4,300,000, wbc. 11,300, eosinophiles 2, juveniles 2, stabs 19, segmented 52, lymphocytes 24, and monocytes 1 per cent. Fasting blood sugar was 105, blood chloride 612, N.P.N. 35.2, creatinine 167, urea nitrogen 15.0 mg. per 100 cc. of blood. Spinal fluid: color clear, pressure 14 cm., polys 2, lymphocytes 1, protein 252 mg., the gold curve 1111232110. The Wassermann was negative.

Course in the Hospital: During the first 24 hours the patient had a thoroughly miserable time in spite of frequent injections of codeine and demerol; 100 mg. of demerol would give him partial relief for approximately 2 hours, when he would again complain of severe pain. On the third hospital day the patient was started on ACTH, 10 mg. in 5 per cent glucose solution, in a very slow drip. We attempted to give this over a 20-hour period. This was repeated daily for 3 days, then it was reduced to 7.5 mg., 5 mg., and 2½ mg. respectively for the next three days, at which time it was discontinued and cortone was started, 25 mg. four times a day by mouth.

Within a few hours after administration of ACTH, the pain was markedly relieved, and the patient was comfortable with only 100 mg. of demerol in each 24-hour period. During the fifth day, when the patient received only 2½ mg. of ACTH, and on the following day when he was on cortone, his pain again became severe. ACTH was begun again, 10 mg. daily, and was continued for the next five days, reducing the dose to 5 mg. after the third day. On the sixth day in the hospital, three days after ACTH was started, the patient developed a total paralysis of the right sixth nerve and experienced some respiratory difficulty.

On the 10th day in the hospital, the patient developed a rather typical toxic psychosis. He became confused, disoriented, delusional, and hallucinated. His nights were restless, he would constantly call for the family, he would think the hospital on fire one minute and a little later that some other catastrophe was threatening.

ACTH was discontinued on the 13th hospital day, and the patient was started on cortisone, 25 mg. q.i.d.; three days later this was reduced to 25 mg. t.i.d. and gradually reduced to 25 mg. b.i.d. Following the discontinuance of ACTH the second time, the psychosis cleared up almost immediately, and the patient experienced little pain. Within a few days he was able to be out of bed and to move around the room. The patient left the hospital on the 33rd day, able to walk short distances. The diplopia had completely disappeared, and, aside from showing extreme fatiguability and weakness, he was comfortable. In spite of the clinical improvement, the spinal fluid protein (252 on admission), which had dropped to 196 on the 11th day, was up to 270 mg. on the 23rd day—10 days before dismissal.

Six months later this man was examined in the clinic and showed no changes in sensation or reflexes. He was still complaining of some weakness. He stated that he could work in the field running a tractor quite comfortably for half a day but became very tired if he attempted to work longer.

CASE 2

Mrs. F. W., a 52-year-old housewife, was referred to this hospital on December 3, 1952, by Dr. A. C. Eitzen of Hillsboro, Kansas. The patient's chief complaint was numbness and weakness in all four extremities, with onset approximately 5 weeks before admission. She had been doing considerable work, redecorating her house, reaching under cabinets, and climbing on ladders. Following this, she noticed some stiffness and soreness in her back between the shoulders but ascribed it to the exercise.

About three weeks prior to admission, while she was lying in bed, her feet felt "like there were big crusts on the bottom." This numbness gradually increased until she had little feeling in her feet, and the same symptoms started in her hands. She also developed a marked weakness in her feet and legs and was unable to stand alone. About ten days prior to admission, she developed a diplopia on looking to the right. Dr. Eitzen reported, the day before her admission to the Halstead Hospital, that the spinal fluid protein was 98, cell count 0, Wassermann negative.

The past and family history were consistently negative. She had always been healthy, had had four normal deliveries and no serious illness or surgery.

Physical examination revealed a rather obese white woman, apparently clearly oriented and free of any mental symptoms. Blood pressure was 120/80, pulse 80, temperature normal; heart, lungs, and abdomen were negative. Pupils were equal, regular, and reacted to light and accommodation, and the fundi were negative. There was a definite weakness of the sixth nerve on the right. Facial muscles were normal, the tongue was normal, speech clear. There was generalized weakness of all four extremities; abdominal reflexes were absent. The patient was barely able to lift her heels off of the bed and was totally unable to stand. She could not feel or identify objects held in her hand and recognized only rather deep pin pricks over the hands and feet as painful. She did complain bitterly of considerable pain throughout the entire region of the thoracic spine and the lower cervical region. This was not aggravated by movement, and she insisted that she had not been relieved by analgesics.

Laboratory Findings: Urine was negative. On admission hemoglobin was 76 per cent or 13 gm., rbc. 3,760,000, wbc. 12,000, eosinophiles, 2, stabs 8, segmented 55, lymphocytes 33, monocytes 2, pathological 0. Liver function as determined by the hippuric acid test was 97 per cent. Kline was negative. Spinal fluid: lymphocytes 4, polys 0, protein 78, gold curve 0112443210. Fasting blood sugar 78 mg., N.P.N. 35.0, creatinine 1.75, urea nitrogen 16.0, chloride 595 mg. per 100 cc. of blood. Sedimentation rate 29 mm. in the hour; 31 mm. 12 days later. Sixteen days after admission, the spinal fluid pressure was 24 cm., lymphocytes 2, polys 0, protein 89 mg. Hemoglobin on the 12th hospital day was 80 per cent or 13.5 gm.

Course in the Hospital: The patient was started on ACTH, 10 mg. in 1000 cc. of 5 per cent glucose, given at a slow rate. This was begun on the second hospital day and continued until the seventh hospital day, when it was decreased to 5 mg. and decreased again to 2½ mg. on the eighth day, after which it was discontinued entirely. The chest x-ray on the 15th hospital day showed some flattening of the left diaphragm with blunting of the costophrenic angle and some tortuosity of the aorta.

For three days after cortone was started this patient continued to get weaker and the sensory loss increased. On the fourth hospital day she was unable to pick up a spoon off her tray to feed herself, but by the eighth hospital day her strength had returned sufficiently so that she could lift her food to her mouth, though she was totally unable to locate objects on her tray by touch. However, during this time the pain was entirely relieved, and she was comfortable. The patient continued to convalesce and left the hospital on the 27th day, barely able to walk to the car.

This patient was seen again one month after dismissal, at which time spinal fluid had a cell count of 10 lymphocytes, 4 polys, protein 35. She is able to do part of her own housework and gets around fairly well.

DISCUSSION

In both of these patients, the acute process of the disease was continuing to progress at the time of admission to the hospital. Both were started on ACTH, which afforded them almost immediate and complete relief from pain. In the first patient, the pain was extremely severe. When ACTH was discontinued and cortisone substituted, his pain recurred. When ACTH was given again until it had to be discontinued because of a toxic psychosis, pain was relieved. In both cases, muscle weakness and sensory loss progressed for at least four days after ACTH was started. Vitamin B complex was given in addition to ACTH and cortisone. The highest spinal fluid protein obtained on one patient was after recovery had begun. Both patients have recovered with no residual symptoms.

Since knowledge of the etiology of this condition is as yet only theoretical, there is no specific treatment. There have been several reports in the literature

of patients treated with ACTH and cortisone; most authors have felt these drugs are of value. Since several of the vitamin B group have proved to be absolutely essential to the nervous system and their absence in itself will produce a nutritional disturbance, practically all physicians give vitamin B and particularly B-12 on an empirical basis. Both of our patients received vitamin B complex by mouth and 30 micrograms of B-12 intramuscularly every third day.

CONCLUSIONS

1. We feel that ACTH was of marked value in offering these two patients complete relief from pain.

2. On the fourth day after treatment with ACTH was begun, both patients began to improve, though previously the disease had been progressing steadily. We have no way of knowing how much the ACTH actually influenced the course of the disease since it is a self-limited illness; we have seen many cases suddenly start improving spontaneously.

3. It is possible that ACTH helped in clearing up both cases with no residual symptoms. However, the convalescent period was approximately the same as in patients we previously treated by other means.

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Home Accidents Cost Money

The financial cost of medical care required as a result of accidents each year in Kansas is, of course, unknown. Some inkling as to the size of the burden might be gained from the following information obtained from Kansas Blue Shield for a few selected types of claims paid during the year 1952, the majority of which resulted from accidents. The payment figures represent actual amounts paid by Blue Shield and not actual physician charges.

CLAIMS PAYMENTS FOR CHILDREN UNDER 19 DURING 1952
BY KANSAS BLUE SHIELD

Diagnosis	*Number*	*Payment*
Fracture of Radius and Ulna	554	$ 22,649.76
Fracture of Tibia and Fibula	155	7,636.16
Traumatic Wounds	2,634	22,403.70
Burns (Emergency Treatment Only)	122	594.50
Diagnostic X-ray (Accident Only)	4,836	31,857.55
Total		$ 85,141.67

The estimated average number of individuals under 19 years of age covered by Kansas Blue Shield during 1952 was approximately 99,400. This represents about one-sixth of the population of Kansas in this age group. If the childhood population of Kansas not covered by this insurance plan experienced accidental injuries of this degree of severity at a comparable rate, the cost to parents for partial physician fees alone would approximate $520,000 for just these few selected types of non-major injuries in one year.

Committee on Child Welfare

PRESIDENT'S PAGE

DEAR DOCTOR:

This will be necessarily brief as your new president is in a considerable state of confusion. (This may be the understatement of the year.)

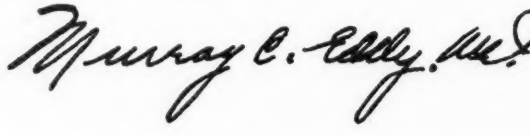
Committee appointments are practically complete, but the year's program can hardly be outlined until after the American Medical Association meeting in June. It is my understanding that several issues should be decided at that meeting which directly affect any legislation we might be considering, and our course cannot be set until later.

I would like to pay a tribute to Dr. Lucien R. Pyle, whose devotion to Kansas medicine, whose calm judgment of controversial issues, have been an inspiration to me and the other officers of the Society. The groundwork done by him and those before him will continue as before to point the way to better medical citizenship, constantly improving medical care for our citizens, and a closer medical community.

I would appreciate personal letters of suggestion from any of you, and I will undoubtedly ask for much advice from all officers, councilors, and committee members as well as the membership at large.

I shall endeavor to be a conscientious servant of the Society.

Yours,



EDITORIAL COMMENT

Veterans' Affairs

At a meeting in Denver on Friday, March 19, representatives of the American Medical Association presented and reaffirmed their stand on medical care for veterans. These representatives made it clear that the A.M.A. places discharged veterans in two separate classes. First are those with service connected disabilities, and second are those with non-service connected disabilities.

Service connected disabilities are those incurred or aggravated in military service, or those suffered following military discharge which are a result of or attributable to military service. Non-service connected disabilities are those ailments which in no way relate to the veteran's service in the armed forces.

The A.M.A. makes it clear that they are in favor of having the federal government provide every reasonable benefit to veterans who were disabled as a result of their military service. On the other hand, they believe that the veteran who suffered no physical or mental mishap while in military service should obtain and provide for his own medical care as does any other individual.

Since this is the stand of the medical profession, the A.M.A. believes we must continue not only to answer questions in regard to non-service connected cases, but we must repeatedly confirm our stand on the government's responsibility to those with service connected disabilities, as it is at this point where many fail to understand our policy. The A.M.A. is in accord with the provision that the government take care of service connected disabilities, and it has cooperated and will continue to assist in providing such care.

For the purpose of this summation, however, let us consider those who are non-service connected cases or those who, when discharged from service, were in a state of physical and mental health as good as or better than when they went in.

A brief history of the medical care program describes the tremendous growth of the Veterans Administration program to veterans with non-service connected ailments. In 1926, just two years after veterans with non-service ailments were receiving treatment for diseases or injuries not incurred in service, 17 per cent were in this class. Two years later, 49 per cent of admissions were non-service connected. In 1931, the non-service cases accounted for 71 per cent of the admissions. By 1944, the sum of $218,000,000 had been spent for veterans' hospital construction, and during that time 80 per cent of admissions were treated for conditions not related to military service.

The Veterans Administration's own figures for the fiscal year 1951 show about 85 per cent of the patients were treated for non-service connected disability, which means 85 per cent of the more than half a million patients.

It is obvious, therefore, that we have had more than adequate facilities to take care of veterans with service connected disabilities for some time, which makes it equally obvious that much of the amount already spent for veterans' care, as well as anticipated expenditures, is for non-service connected cases. There is little need of mentioning that both the number and percentage of non-service connected cases rise steadily. Consider the impact of the basic trend by realizing that by the year 1970 there will be more than three times as many living veterans over 45 years of age as there are today and more than 10 times as many living veterans over 65.

The Veterans Administration now has a total of almost 120,000 hospital beds and is spending more than $600,000,000 annually on its program. In 1945, General Frank T. Hines, administrator of Veterans Affairs, said that by 1975 a fair estimate of hospital beds for veterans would be 400,000 with an annual expenditure of $1,000,000,000.

The A.M.A. urges that the federal government discontinue the care of non-service connected cases except for war veterans with tuberculosis and those with mental or nervous disorders who are unable to pay for their own hospital care, and that even this temporary exception be made until local facilities are adequate to handle such cases.

The country must decide the basic question of whether the cost of the present and future program is a justifiable burden on the taxpayers of this country. The consideration must be studied with concern for the entire population and not a particular segment.

There are many reasons for the A.M.A. stand on this subject. The artificial shortage of doctors created by heavy demands on the medical profession by the government creates a public relations problem sometimes difficult to explain. This same demand also creates shortages in allied personnel, which necessarily means higher wages to attract nurses, technicians, etc., to civilian hospitals. This naturally creates higher prices for the civilian consumer of hospitalization.

The average hospital stay in a Veterans Administration hospital is 30 days, compared to 7½ days for a person with similar disability in a civilian hospital. Many times this brings the over-all cost of illness higher in a VA hospital than in a civilian hospital, even though the medical care is secured without charge. Loss of time at work and travel expense are among the governing factors in this observation.

Today there are about 20,000 veterans waiting to get into veterans' hospitals, of which only three have service connected disabilities. This substantiates the fact that additional hospitals which are being built are constructed specifically for the purpose of taking care of non-service connected cases. All of these factors and many more are important in considering the over-all picture of veterans' medical service.

The most important question the American people must face today, however, is one of American principle. Should veterans who come out of uniform healthy and unhurt be absolved of all personal responsibility for their own health care? Should non-veterans continue to assume responsibility for their own care in addition to taking on additional tax responsibility to pay for a gigantic veterans' medical care program? The answer is found in studying the basic American principles involved.

Orgone Energy

Have you ever heard of orgone energy? Probably not. But perhaps you have patients who believe that it possesses properties that will prevent and cure a variety of serious diseases, including cancer.

Physicians, of course, can spot a medical "phony" immediately, and they may believe that the lay public is discerning enough to recognize and reject impossible claims. However, with science making daily announcements of new discoveries, new "miracles," it is not surprising that some are deluded by false hopes in health fields. Orgone energy is a case in point.

It was in 1939 that Dr. Wilhelm Reich, who once was associated with Dr. Sigmund Freud in Vienna, entered this country and devoted his energies to the invention and production of an "Orgone Energy Accumulator." It was promoted through pamphlets carrying such titles as *The Discovery of the Orgone, Cosmic Superimposition, People in Trouble,* etc.

The most widely distributed model is a cabinet a little smaller than a telephone booth, constructed of alternate layers of wood, metal, glass, and steel wool, with no electrical or other energy connections. This box is supposed to accumulate the "orgone energy" from the atmosphere. The patient sits in the box, and allegedly stored up energy enters his body and cures his ailments. Other models, based on the same accumulation theory, are the "Shooter Box," "Orgone Energy Blanket," and "Orgone Energy Funnel."

You think no one would be gullible enough to place confidence in such a device? The Food and Drug Administration, United States Department of Health, Education, and Welfare, estimates that more than 1,000 were sold or rented at prices ranging from $15 to $225 each.

No further threat exists as far as orgone energy is concerned. The Food and Drug Administration took action in March to bar such devices from interstate commerce. The decree also bans distribution of claims that orgone energy exists and calls for the destruction of all promotional material containing instructions for the assembly and use of the enjoined devices. But the problem remains—the public must be protected from the perpetrators of such sales.

It is interesting to note the reaction of the discoverer of orgone energy to the injunction. When the action was filed, Dr. Reich published a response in which he stated, "Inquiry in the realm of Basic Natural Law is outside the judicial domain, and I shall not appear in court as the 'defendant' against a plaintiff who by his mere complaint already has shown his ignorance in matters of natural science."

The following comment was made by the Commissioner of Food and Drugs, Charles W. Crawford: "Dr. Reich has long contended that only the hopelessly ignorant could disagree with his theories or doubt his miraculous cures with Orgone Energy Accumulators. Repeated challenges were issued in literature, widely distributed by the Wilhelm Reich Foundation, daring medical researchers and physicists to test accumulators adequately. FDA accepted the challenge and has concluded that there is no such energy as orgone and that Orgone Energy Accumulator devices are worthless in the treatment of any disease or disease condition of man."

This particular issue is unimportant to Kansas physicians. It serves only to stress the fact that the public must be safeguarded in the field of health, where irreparable harm may result to persons who abandon or postpone the usual forms of medical treatment while pinning their faith on worthless devices.

Honor in the Practice of Medicine

Much has been said of late to the effect that the physician of today does not occupy the same position of influence in the community that he occupied a few decades ago, and that he has lost dignity in the estimation of the laity. It has also been claimed that even "modern medicine, with all its advantages, improvements and enlarged resources" stands before the public shorn, apparently, of much of that dignity and appreciation that was formerly extended to a less developed science. That this is true in some measure cannot be denied; but it is doubtless due to the commercial spirit of the age in which we are living. The change in social position is due largely to the changes that have taken place in the people. A half century ago the interests of the country were less diversified, and its wealth less developed and distributed.

Many of the luxurious habits of today were un-

known to public and private life. Then education and character were quite universally regarded as the highest measures of worth, and counted for more than wealth; but today the influence of wealth and the commercial activity of the country seem to overshadow and detract attention from the more quiet channels of life in which the physician, from necessity, must have his sphere of activity.

I do not share the pessimistic view that the profession today, as a whole, is unworthy of the high regard and confidence that was once reposed in it by the public. To the contrary, if education, morality, a high type of citizenship and a sacrifice of self in undeviating devotion to duty were accepted as the measure of true worth, no calling in the land would occupy a more lofty height on the pinnacle of worldly esteem than the medical profession of today. However, there are some among the physicians of today who are not faultless, but whose misguided conduct fails to merit either our indulgence or our approval. It is to be regretted that the tendency on the part of some of the well-meaning members of our profession is to disregard the traditions of the profession and to give themselves over to the commercial spirit of the times which is, alas! too common in many walks of life.

The above is a portion of the president's address made before the Kansas Medical Society by J. E. Sawtell, M.D., Kansas City, Kansas, in May, 1908.

Blue Shield Appointment

The appointment of Thomas R. Reed, Topeka, as physician relations representative for Kansas Blue Shield was announced last month by Proctor Redd, director of professional relations for Blue Cross-Blue Shield. Mr. Reed began his new duties May 1. He has been an enrollment representative of the plans since 1951.

Student Center Opened at K.U.

The new $600,000 student center at the University of Kansas Medical Center was officially opened after dedication ceremonies held on Sunday, April 4. The building houses the Battenfeld Memorial Auditorium and the C. B. Francisco Memorial Lounge.

Among the speakers taking part in the dedication were Dr. W. Clarke Wescoe, Dr. Franklin D. Murphy, and Dr. Galen M. Tice.

The auditorium is the only one at the center that can accommodate the entire student body and faculty simultaneously, 924 seats. It is equipped with theater seats, theater lighting, staging, and projection booth. It was constructed and completely furnished by a gift from the Battenfeld Memorial Fund established by Mrs. Edward H. Hashinger in memory of her son, Dr. Jesse R. Battenfeld, Jr., who died in 1945 in a military plane crash.

The auditorium will be used for student assemblies, postgraduate courses for physicians, student-faculty meetings, and for public information and service programs.

In addition to the lounge, auditorium, and dining room which seats 125 persons, the center also includes a book store for medical texts, a snack bar and a recreational library on the first floor. A seminar room, recreation room, showers, and storage rooms are in the basement. The second floor contains 13 double hotel-type rooms for visiting lecturers and participants in postgraduate courses.

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SERVICE SEPARATIONS

As a service to physicians and communities in this state desiring additional medical personnel, the Journal of the Kansas Medical Society will publish in this column each month the names of medical officers who will shortly be separated from the armed forces. These are men who volunteered from Kansas, and many of them will probably be interested in finding locations in this state. Anyone interested in contacting these physicians may write to the address here given.

Theodore L. Batchelder, M.D.
Garden City, Kansas

Albert I. Decker, Jr., M.D.
826 Alabama Street
Lawrence, Kansas

Lyle Harris Edelblute, M.D.
1210 Thurston Street
Manhattan, Kansas

Wendell L. Good, M.D.
Perry, Kansas

Simon E. Hershorn, M.D.
Wesley Hospital
Wichita, Kansas

J. Gilleran Kendrick, M.D.
4224 East English
Wichita, Kansas

Kenneth L. Knuth, M.D.
Atwood, Kansas

Francis X. Lenski, Jr., M.D.
703 South Cottonwood
Iola, Kansas

Stanley R. McEwen, M.D.
447 North Belmont
Wichita, Kansas

Clinicopathological Conference

CASE PRESENTATION

Dr. Paul Sherman, resident in surgery: This is a 64-year-old white man who was admitted on the surgical service December 4 and died five days later. When he entered, he was not oriented to time, place, or person. He was an obese, hot, and dry male with tachycardia and labored, fast respirations.

The history itself, not obtainable from the patient, was given by his son. Apparently it began November 26, after the Thanksgiving Day dinner. The patient became nauseated and had abdominal distention and discomfort. The symptoms progressed. He was taken to his local hospital where he was treated with gastric suction and intravenous fluids. His symptoms of abdominal distention, fever, and malaise continued and, just prior to his admittance here, he became disoriented.

The past history revealed the patient had always been a healthy, hard working, heavy-eating man who had never before been hospitalized. He had had typhoid fever at age 18 and had had mumps as an adult. He had had an attack similar to his present illness three years ago and was said to have been jaundiced for three weeks. During this illness his weight decreased to 180 pounds but thereafter returned to 225 pounds, his approximate weight at time of admission.

On review of systems it was thought that perhaps he had precordial pain with severe exercise, but this was not clear. Genitourinary history revealed a nocturia of one to two times a night and a decrease in the size of the urinary stream.

The family history revealed that one sister died at age 68 of tuberculosis. One sister died at age 70 of heart disease. His father died of a stroke. His mother had hypertension.

The physical examination revealed a disoriented, elderly, obese white male with florid facies, hot dry skin, and dyspnea. He volunteered no complaints. Temperature was 103.0 F. Pulse was 120 per minute, regular. Respiration was labored, 24 and 28 per minute. Blood pressure was 120/80. Body weight was 224 pounds with height 68 inches. Oral mucous membranes were dry; there was no rigidity of the neck; eye, ear, nose and throat examination was unremarkable.

Examination of the thorax showed a moderately increased AP diameter plus obesity. Respiration was diaphragmatic in character. Percussion notes were resonant throughout; although the diaphragms were high, they were mobile. No rales were heard. The heart sounds were faint without murmurs or thrills. There was a tachycardia. The peripheral vessels did not show undue distention.

The abdomen was obese and moderately distended with moderate tenderness in the epigastrium to deep palpation. No rebound tenderness could be elicited. No bowel sounds were heard; fluid or masses were not palpated in the abdomen. Percussion note was tympanic over the entire abdomen. The rectal examination showed benign prostatic hypertrophy of Grade I to II; otherwise the rectal examination was negative. Extremities were normal. The neurological examination was not significant.

Laboratory examination of the urine showed it to be acid in reaction, 1.022 specific gravity, heavy trace of albumin, some white blood cells, and numerous crystals. Remaining urinalyses during his hospital course were obtained by catheterization and showed many red blood cells, 10 to 20 white blood cells per high power field, one negative urine culture, and on three different occasions a specific gravity of 1.010. The blood studies showed hemoglobin of 14.7 grams (90 per cent); 5.3 million red cells; 27,000 to 34,000 leukocytes with an increase in the polymorphonuclears and from 2 to 21 non-filamented forms in repeated examinations. Wassermann and Kahn were negative. Serum calcium and phosphorus was 3.3 and 5.9 mEq./liter respectively on admission, and 4.6 and 6.5 respectively the day he died.

On December 5, 1953, serum amylase was 64 milligrams per 100 milliliters, and serum lipase drawn the same day and reported on the day of death was 1.22 units. On admission, the NPN was 116 and rose to 138 the day of death. Creatine was 6.7 and reached a maximum of 8.0, December 7, 1953, and 6.0 on the day of death. Blood sugar was 155 on admission. Serum sodium was 151 mEq./l. on admission and 159 the day of death. The CO_2 was 20 mEq./l. On December 7, 1953, the blood pH was 7.37. Urine and gastric suction contents were analyzed for sodium, potassium and chloride to aid in maintaining proper electrolyte balance. Serum agglutinations for typhoid O and H, parathyroid A and B, and Brucella were all negative.

Hepatogram on December 7, 1953, showed a direct bilirubin of 0.7 mg. per 100 ml., a total of 1.0 mg. per 100 ml. Alkaline phosphatase was 1.5 millimol units. The cephalin flocculation was 3 plus. Thymol turbidity was 4, serum albumin 3.0, globulin 2.0, total cholesterol 175 mg. per 100 ml. with 50

From the University of Kansas Medical Center. Edited by Glen R. Shepherd, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, pathology, radiology, and surgery, and the third and fourth year classes of medical students.

per cent cholesterol esters. A blood culture on December 6, 1953, was negative. The report was postmortem.

While in the hospital the patient was treated with intravenous dextrose, normal saline, potassium chloride solutions, Miller-Abbott tube intestinal suction, streptomycin, and penicillin therapy intramuscularly. Special sigmoidoscopy was done immediately upon admission and was found to be negative to 27 centimeters. The medical consultant added to the history that the patient's onset of illness was accompanied by cramping abdominal pain.

The temperature showed a drop to normal during the first 24 hours with an immediate rise again to 103° F. by the end of the first hospital day, and thereafter a gradual decrease to 102° F. throughout his stay. The pulse rate decreased and remained about 90 through this period. Respiratory rate continued to be rapid and labored, 36 to 40 per minute. By the second hospital day, the urine output was 1700 cc. and remained good thereafter.

On December 6, 1953, the patient began to pass gas and some loose watery stools of brown color. On December 7, 1953, he had multiple green stools.

During his hospital course his blood pressure was maintained within normal limits until his death. During that day it had been approximately 140/80 with pulse rate of 96 and agonal temperature of 102.6°.

Dr. Delp, chairman: Are there any questions concerning this history?

Q: Were the stools the last two days still multiple and green?

Dr. Sherman: Yes, small in amount, multiple, green, about 5 to 15 a day.

Q: Do you have any more information about the severity of the pain experienced on Thanksgiving Day?

Dr. Delp: He apparently did not consult a doctor on that day. He didn't consult a doctor until two or three days after that.

Dr. Sherman: That's right, he consulted a doctor several days later at the insistence of his family.

Q: Do you know anything about his past history before this present illness and what was his urinary output during the time that he was in the hospital and preceding the hospitalization?

Dr. Sherman: We're not sure about the past history. Hypertension was suggested during two talks with his son. His urine output was low on the day of admission. It was 500 to 700 cc. With intravenous fluid administration, it became 1700 cc. the second 24-hour period and thereafter about 1700 cc. to 2000 cc. per 24 hours.

Dr. Delp: Any other questions? If not, we have some x-ray films as well as an EKG. Will someone put on the EKG for us?

Mr. John Gorman, fourth year student: The first tracing on this patient was done December 3. Another was done on December 8. This pattern would be that of tachycardia with left ventricular strain. It is also the pattern you see in hypopotassemia, with the lowered T waves, the long swooping RST segments and the increased QT. The December 8 tracing is normal.

Dr. Delp: Relihan, let's see the x-rays.

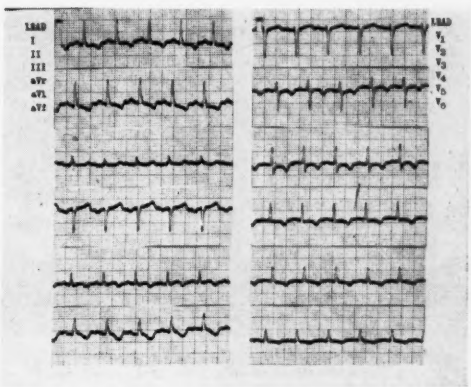

Figure 1. Admission EKG, December 3, showing sinus tachycardia, T waves inverted in leads I, II, aVF, and V_2 through V_6, increased QT and long RST segments indicating left ventricular strain and hypopotassemia.

Mr. Donald A. Relihan, fourth year student: We have two films made on the day of admission, the 4th, one chest film, and one flat plate of the abdomen. The chest film was portable with the patient poorly positioned. Possibly some of our interpretation would be invalidated because of that. The positive findings are elevated diaphragms and soft diffuse infiltrated lung field. I think this possibly could be interpreted as pneumonia.

The flat plate of the abdomen shows dilated gas-distended sections of large and small bowel, with a stepladder pattern of the small bowel in mid-abdomen. Barium enema study on the next day, the 5th, shows dilated proximal portion of the colon. The distal colon is not dilated, possibly due to the fact that he had been previously decompressed by enemas. The widely dilated gas-distended small bowel is still apparent. This is a post-evacuation film which shows retention of the barium in the colon. This pattern is what I would interpret as adynamic ileus without evidence of obstruction at any point, and the pattern of the colon is normal. The next film was made on the 8th, the day before death and in this film we can see the Miller-Abbott tube. There has been some decompression, although it is possible to see the small bowel in some portion of this film still distended by gas.

Dr. Donald R. Germann, assistant professor of radiology: I might add one point. I think the stu-

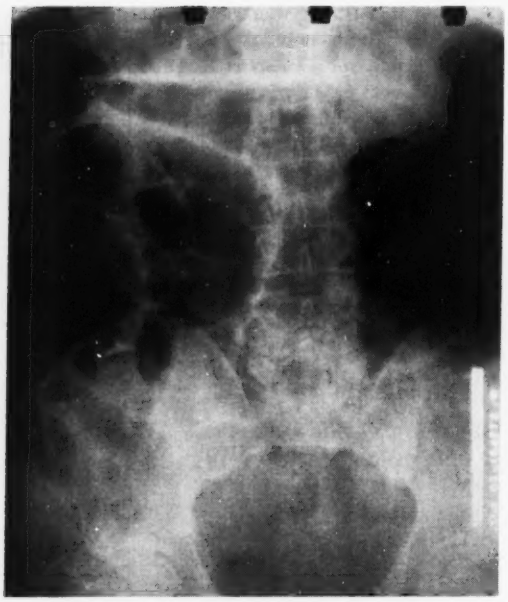

Figure 2. Flat plate of abdomen made December 4 showing dilated gas-distended sections of large and small bowel with stepladder pattern of the small bowel in mid-abdomen.

dent's interpretation of ileus was good, but one thing that concerned us which didn't come out was that this enema was done during the night. The next morning when we looked at our film we were concerned by certain shadows we saw. It looked like some barium might have leaked out of the sigmoid into the peritoneal space. We were afraid that we had actually perforated the sigmoid. That was the reason for this film which was taken the next morning. There was about 12 hours difference. No evidence of perforation appeared. We were satisfied in our own minds that this was not a perforation, but it was one of the things that went through our minds.

Dr. Delp: Any further questions about the x-rays? They might have some importance to the solution of the problems. It might seem that this is a rather straightforward case, a clinical entity that should be immediately recognized, but I'm sure that that is not so.

These things have interested me. The pain must have been rather mild at onset. At least we can grade it by saying that the patient was not forced to call a physician. It was only after two or three days of illness that he did finally see a physician. He was evidently quite ill then, because hospitalization was recommended immediately and he subsequently was transferred here for further care.

I think we must answer the questions as to whether or not true intestinal obstruction or simply an adynamic ileus, as Relihan has indicated, was present. I would like to hear some explanation for the patient's convulsive disorder which he apparently had the last several days of his illness. The blood chemistry changes also need explanation. Mr. Smith, may we have your differential diagnosis?

DIFFERENTIAL DIAGNOSIS

Mr. Wallace B. Smith, fourth year student: The case for today presents, as Dr. Delp has said, less of a problem right now than it did when I first read the protocol. I think in any discussion we should always try to tie as many things to one disease state or one diagnosis as possible. We should always look for the simplest explanation of a patient's illness.

The patient, if I might review a little bit, had pain in his abdomen. He had nausea, vomiting, and distention. In addition to this, he had persistent fever together with an increasing amount of disorientation. On admission to this hospital, he had a distended abdomen. It was mildly tender, especially in the epigastrium. He had a low, fairly fixed specific gravity urine with some crystalluria and some pyuria. He had a leukocytosis, a low calcium, and a high phosphorus. He had an increased sodium, a decreased chloride, a hyperglycemia and an azotemia. He was treated vigorously with fluids, antibiotics, and other accepted treatments including electrolytes. They were analyzed carefully to determine just exactly what he needed. In spite of this, he continued to worsen and die.

It's a toxic type of manifestation all the way through with a fever and leukocytosis. I think that we are obligated to explain these facts. I think the principal concern we have here, at least when he came into the hospital, is why he was distended and why he had an ileus. I'm not going to go through all the causes that are possible for an intestinal obstruction or an ileus, but I would like to include some of the things suggested by this history.

First of all, the man had a sister who died at age 68 of tuberculosis. Tuberculous enteritis often produces a type of colicky pain with an ileus which may or may not be present. The x-ray picture should show some evidence of ulceration, or we should have some pulmonary signs of tuberculosis or some clinical history suggestive of tuberculous process. We have none here.

Second, it could be neoplastic, either benign or malignant. These things may be asymptomatic until they produce the ileus. However, the x-rays cannot point to anything which we can interpret as a constrictive neoplastic or a space-taking neoplastic lesion.

Third, he could have an obturation of the small bowel, and there are several things that cause this. The only thing I think we need to think of here is possibly a gallstone obturation due to a choledocho-

or cholecysto- duodenal fistula with a possible gallstone in the gastrointestinal tract. The man has a past history of similar abdominal pain with some jaundice. We can assume that the man had cholecystitis or cholelithiasis. He's fat and a heavy eater.

Fourth, the man could have a volvulus. This shows up usually in men past middle age. At least it is seen more frequently in males in late middle life. It frequently follows ingestion of a heavy meal. These people are seized with abdominal pain, usually quite severe, and it's accompanied by more or less distention, depending on where the volvulus occurs. In the sigmoid area, it can produce a great deal of distention. I think the fact he had a sigmoidoscopy shortly after entering the hospital suggests perhaps that was the admitting diagnosis. However, I think that it was pretty well ruled out by 27 cm. of negative sigmoid and no evidence of a volvulus by x-ray.

Another cause of an adynamic ileus that we have to entertain is acute cholecystitis, either by itself or with a possible rupture of the gallbladder, with a bile peritonitis developing. This man had a history suggestive of cholecystitis or cholelithiasis, and I think that we can't rule it out on the basis that we have here. This man had an ileus, and it could have been on the basis of a gallbladder rupture or acute cholecystitis. The pain is not typical, and the man didn't look like he had peritonitis, at least unless he was so foggy that he just couldn't respond at all. I don't believe he was comatose. I think that pneumonia and empyema can cause an ileus with a diaphragmatic pleurisy. Although he had a leukocytosis and a fever, he didn't have the other signs that go with pneumonia. Uremia by itself can cause an ileus. I'm not sure of the mechanism of this ileus, but it has been reported as being seen and not too infrequently in uremia.

A disease that frequently can produce a similar picture to the one we have here is acute pancreatitis. The onset of acute pancreatitis is with sudden severe abdominal pain. It usually radiates to the left back underneath the scapula. The patient usually goes into shock. We don't have that history here.

There is a certain percentage of cases in which this is not seen, and the patient may have just colicky pain. Also we have other evidence of acute pancreatitis, I think. On admission, a low calcium and a high phosphorus were found. It's the sign of pancreatitis with fat necrosis and calcium deposits which show up in the omentum and peritoneum at exploration. Also the loss of calcium soaps through the gastrointestinal tract from a lack of pancreatic enzymes produces a low calcium, and you find a reciprocal high phosphate value. He had a hyperglycemia. This could be accounted for by the destruction of pancreatic tissue.

The amylase and lipase reports that we have are not startling. The amylase was within normal limits, but we would not be concerned about that because we didn't see him when he first had this attack. The amylase could long since have become normal again. The lipase is elevated only slightly, and I think that also goes along with a gradual decrease in the lipase value as either destruction of pancreatic tissue or some other mechanism occurs which lowers the lipase. It usually stays up for only 10 to 14 days after the attack of acute pancreatitis.

He had an increased white count with fever. This could be an infectious process going on in the pancreas. He had a history of previous gallbladder disease, and that is often seen in patients who develop acute pancreatitis. He had slightly increased serum bilirubin. He had shaking and convulsions which developed terminally. This could be explained by the low calcium. He had epigastric tenderness, and he had a terminal diarrhea which is often seen in cases of acute pancreatitis that terminate fatally. The last two or three days are often characterized by diarrhea.

The other thing which concerned me in this protocol was the increasing azotemia. Certainly, with the disorientation that the man had, I think we can say that he had uremia. Acute pancreatitis can produce lysis of viscera inside the abdominal cavity with areas of either necrosis or gangrene and associated with elevation of the NPN. So I think that I'll hang my hat on acute pancreatitis with secondary uremia and death in this patient due to uremia.

Dr. Delp: Thank you, Smith.

CLINICAL DISCUSSION

Dr. C. Frederick Kittle, assistant professor of surgery: All the interesting things that were pointed out are in retrospect, with regard specifically to renal function, the specific gravity at the time of admission in a patient who was febrile, who was dehydrated, and whose urinalysis also showed a heavy trace of albumin. I think it would indicate that he did have a considerable degree of renal insufficiency at that time.

If we pursue the urinalysis a little further, we notice that there were always blood cells present, even in amounts up to 10 to 20 white blood cells per high power field. This is much more than should be present on the routine urinalysis. Particularly with such a large number of cells per high power field, one would have to say that the urine was infected.

Along with the renal aspects were the hypocalcemia and elevated phosphate. He was seen by Dr. Valk, who thought that he had an appreciable degree of renal insufficiency because of these facts that I have just enumerated, and who directed attention to increasing the serum calcium by continuing the administration of calcium gluconate.

If we turn aside from the renal aspects for a moment and consider the entire problem this man presented, we have a rather long case. For 9 or 10 days there's a patient who had a persistently febrile course with abdominal symptoms and abdominal findings on examination, characterized by nothing specific as far as any one diagnosis is concerned. Consideration was given to the possibility of pneumonia when he first entered because of the appearance of some rales in his chest. The site of these was noted on the x-ray examination. As you are aware, an ileus can occur with severe pneumonia, and this is one of the initial suggestions made in this particular case. His subsequent course could not bear this out.

The next possibility that suggested itself by the prolonged distention was that of intestinal obstruction either of the small intestine or of the large intestine. He was sigmoidoscoped immediately on admission with negative results. The fact that he had bowel movements and passed gas toward the end of his hospital stay would be evidence, I think, that any obstruction was not a complete one.

So I think we should turn from the diagnosis of an obstructive lesion to some sort of inflammatory lesion within the abdomen. The diagnosis of pancreatitis is the diagnosis that answers all of these criteria as well as fitting in with the various laboratory results that were obtained. The lowered calcium in cases of acute pancreatitis is something that perhaps is ignored too frequently.

Actually, what happens is that with the release of the trypsinogen that becomes activated into trypsin and the subsequent digestion of the pancreas with the break through of the pancreatic enzymes into the abdominal cavity, lipase is released. Lipase then proceeds to break down the fat, the omentum, and the mesentery with the subsequent formation of a large number of unsaturated fatty acids. These unsaturated fatty acids react with the calcium and the serum with the formation of soap in the mesentery and in the omentum and also a marked hypocalcemia. This patient first began having irregular twitching episodes on the third day before death and continued to more generalized convulsions on the day of his demise.

Regarding indications for operation in this particular patient, I think that perhaps some difficulty was encountered because the patient had been sick for almost a week before he was admitted to this hospital. If he had been observed for several days, the fact that his course was becoming worse would in itself have been indication for a laparotomy. At the time of laparotomy, the diagnosis could have been established and therapy indicated. I think it would be better not to proceed with the discussion of therapy in this particular case until after the pathology report.

Dr. Delp: Dr. Allen, would you care to make any comments concerning this patient before we see the pathology report?

Dr. Max Allen, associate professor of medicine: My thought after seeing him was the possibility of pneumonia as the reason for the ileus. I think usually in the patient who has disease above the diaphragm, pneumonia is responsible for ileus. That disease would be fairly obvious. Other causes would include both renal colic and gallstone colic, back injuries, and medications.

I think the tendency for hypocalcemia and convulsions might be aggravated in this patient by uremia and the slight alkalosis which he had originally. This later probably was followed by the acidosis of uremia.

When I first saw him, I talked to one of the relatives, who led me to believe that this man was a stoic individual in that he would tend to minimize his pain or discomfort. I charted this because I thought his general appearance might be one of that sort. Finally, I think that, as someone mentioned, this man very easily could have previously been hypertensive so that any degree of shock which might occur in his early illness would certainly be a factor to be considered in precipitating acute tubular necrosis.

Dr. Delp: The total length of time between the onset of illness and death was about 14 days. Would anyone else like to comment before Dr. Firminger tells us about this case?

Dr. Stanley Friesen, associate professor of surgery and oncology: Although I think the likely diagnoses have been mentioned, no one has mentioned acute appendicitis which, in my opinion in this particular patient, could be a very likely finding—appendix rupture followed by peritonitis.

Dr. Delp: The third year medical student wrote the following diagnostic possibilities on the chart: (1) acute pancreatitis, (2) mechanical ileus due to small bowel obstruction, (3) rule out ruptured appendix with retroperitoneal appendiceal abscess and peritonitis, volvulus, (4) chemical peritonitis, (5) acute diverticulitis, and (6) acute nephritis.

PATHOLOGY REPORT

Dr. Bernard L. Klionsky, resident in pathology: In the abdomen there were extensive areas of fat necrosis throughout the peritoneum, omentum, mesentery, and surface of the diaphragm and extensive swelling, necrosis, and hemorrhage in the region of the pancreas, which was almost completely destroyed.

There was, in addition, a chronic cholecystitis; a small, shrunken, thick-walled gallbladder, which contained one small mulberry-type stone. An additional stone was in that segment of the common bile duct which was shared in common with the

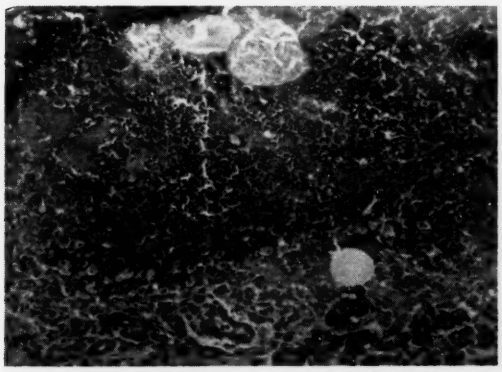

Figure 3. Photomicrograph of pancreas section, showing acute hemorrhagic pancreatitis.

main pancreatic duct. In other words, these two had, for about one centimeter, a common opening to the ampulla of Vater, and there was a small stone within that common channel.

The other main finding was in the kidneys, which were extremely large and pale, weighing over 500 grams. There were a number of small cysts grossly. The diagnosis we made was an acute nephrosis of some kind. Microscopically, we will see exactly what it was.

The heart was enlarged, weighing 450 grams. The other findings, I think, are relatively incidental. There was an ascaris worm in the intestines. In regard to intestinal obstruction, an adhesive band was between the cecum at a point 15 cm. distal to the ileocecal valve. A band was between that point and the edge of the liver, which band had produced a dilatation and a partial obstruction of the proximal cecum. There also were a few diverticuli but no evidence of diverticulitis. Another finding I neglected to mention was a massive partial infarct of the liver, and there were many thrombosed intrahepatic branches of the portal vein. We saw no other gross thrombi.

Dr. Harlan I. Firminger, associate professor of pathology and oncology: Though we looked carefully for thrombi in the pancreatic veins and were unable to see them grossly, microscopically they were apparent. There were organizing thrombi in veins in the pancreas due to inflammatory involvement of the wall of the pancreatic veins. Some of these had given rise to emboli which went to the liver and went into the portal vein in the liver and gave the picture of an organizing embolus in the wall of the portal vein. He did not have bacterial peritonitis with spread up to the lymphatics into the liver.

The kidneys, as Dr. Klionsky mentioned, were large and pale, typical of acute nephrosis. Microscopically, we found evidences of a healing lower nephron hypoxic type of nephrosis with regenerating tubules. Actually, there were a fair number of mitotic figures seen. One area showed a granuloma-like formation which actually was cast material, perhaps of renal epithelial cells which had died and had been sloughed but also albumin and sometimes pigmented material which obstructed the tubule, causing its wall to break down and be extruded into the adjacent tissue. This takes time, and it helped us to date the time at which the actual nephrosis came about.

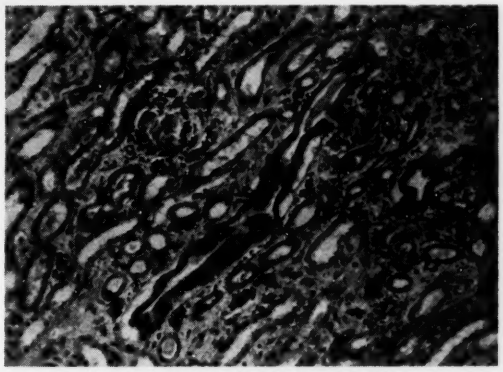

Figure 4. Photomicrograph of kidney section, showing healing lower nephron nephrosis with regenerating tubules and cast material.

Another lesion which one finds in this type of nephrosis is a chronic inflammatory infiltration at the corticomedullary junction about the veins and venules. Here one sees a mass of chronic inflammatory cells projecting into a venule. This is also typical and helped us to date the onset of this lesion.

If I may reconstruct the case as I see it at the moment, it is essentially this. This is a 64-year-old man who had probably liked to eat most of his life. He had some metabolic defect which many people have of putting on fat, and he became obese. Along with this, probably he developed some stones in his gallbladder. Indeed, he had difficulty some three years before. A gallstone of similar type to that found at autopsy produced some symptoms and jaundice and apparently passed without further difficulty.

After eating Thanksgiving Day dinner, the gallbladder was vigorously stimulated by the meal, and another stone was extruded into the common duct but was unable to get through the ampulla. The patient was unfortunate enough to have the anatomic relationship many people have, of the pancreatic duct joining with the common duct about one centimeter before it entered the ampulla and into the duodenum. The stone lodged at this point and produced obstruction. The obstruction produced a reflux of bile perhaps, but at least it increased tension, this produced back pressure in the pancreas, and

there was leakage of pancreatic enzymes out into the surrounding tissue, and an acute hemorrhagic pancreatitis developed.

He must have had severe pain at that time. He must have been a stoic individual. Some older persons do not show much pain, possibly due to elevated thresholds.

In any event, I suspect he may well have gone into shock at that time. Shock for him might have been present with a fair blood pressure. Subsequently he was admitted to the hospital and treated. Almost surely he had oliguria at that time. On arrival here there was evidence of uremia, but the oliguria was beginning to clear. I'd date the acute renal damage 10 to 14 days before death, based on the morphologic changes we see. I think he could have had shock and a hypoxic type of change at the time of the initial onset of disease and that that alone—the prolonged shock for six or so hours, followed by recovery—will produce a lower nephron nephrosis such as we see here and could well account for the renal difficulty.

I think practically all of the laboratory findings which were described can be explained on the basis of the morphologic changes we see. The elevated white count and the renal disease are understandable. I think the phosphorus retention could be explained by the renal damage, and that in itself would tend to lower the calcium, although I think the lowered calcium is also part of the disease in which calcium is taken up with fatty acids. There was some disturbance in liver function. I don't know whether or not that's explained on the basis of the peritonitis.

Dr. Delp: Any questions of Dr. Firminger?

Q: Were any vascular changes noted in the kidneys?

Dr. Firminger: Not particularly.

Dr. Delp: I'd like to have a few comments about the treatment of acute pancreatitis, Dr. Kittle.

Dr. Kittle: I think treatment should be directed in two areas: first the local treatment and then the general treatment. The local treatment should be aimed at those measures which will decrease pancreatic secretion. In this particular patient, that was managed by gastric suction, aspirating the gastric contents so that they did not stimulate gastric secretion. One should also give frequent doses of atropine. The other local measures are those designed to control the ill effects of the enzymes after they have spilled out in the peritoneal cavity. Generally there is peritonitis, so massive doses of antibiotics should be given.

With regard to the general supportive treatment, these patients usually have a reduced blood volume. They go into shock because of the exudation of large amounts of fluid from the mesentery and the peritoneal surfaces. This fluid must be replaced by blood or by plasma. I think that probably this patient may have had a reduced blood volume, even though his blood counts were within normal limits. I think there is every indication that his blood volume was decreased. Supportive therapy has to be considered to maintain normal blood volume. If hypocalcemia develops, repeated injections of calcium gluconate are necessary.

If the diagnosis can be made short of a laparotomy, then it is usually thought that conservative management should be tried first.

If the patient continues to become worse and does not improve on conservative measures, then laparotomy is usually indicated, at which time some sort of surgical drainage of the extrahepatic biliary tract should be attempted. If there are any localized abscesses or localized collections of fluid, for example in the lesser omental cavity or in the peritoneal cavity elsewhere, those are drained. There is no really satisfactory treatment other than drainage of the extrahepatic biliary tract and any abscesses.

A serious implication of acute pancreatitis is recurrence of the attacks and possible development of chronic pancreatitis.

ANATOMICAL DIAGNOSIS

Primary

Chronic cholecystitis and cholelithiasis, with two small mulberry stones, one at the ampulla of Vater beyond the confluence of the common bile duct with the duct of Wirsung.

Dilatation of the common bile duct, slight.

Acute hemorrhagic pancreatitis, advanced, with fat necrosis involving the mesentery, omentum, parietal peritoneum of the diaphragm, hilus of the left kidney, and periadrenal fat on the left (history of onset of abdominal discomfort and distention 14 days before death).

Organizing thrombotic occlusion of pancreatic vein.

Organizing emboli occluding intrahepatic branches of the portal vein.

Recent massive incomplete infarct involving both lobes of the liver.

Acute passive congestion of lungs, slight, and of liver, moderate.

Acute hypoxic nephrosis (history of uremia and severe electrolyte imbalance for five days before death).

Focal submucosal hemorrhages of the rectum.

Fibrous adhesions between the midportion of the ascending colon and the liver with retrograde dilatation of the colon.

Atelectasis of the posterior basal portions of all lobes of the lungs.

Diffuse emphysema of remaining portions of the lungs.

Acute hyperplasia of the spleen, slight.

Focal depletion of lipoid in the cortex of the adrenal glands.

Acute ulcer of the stomach and acute ulcerative esophagitis (history of repeated emeses beginning 14 days before death and of intubation for 8 days).

Acute hemorrhagic cystitis and urethritis (history of indwelling catheter for 5 days before death).

Recent healing linear incisions in the left femoral regions and above both medial malleoli, multiple puncture wounds on forearms and dorsum of hands (history of intravenous injections for 8 days before death).

Segment of Miller-Abbott tube in ileum.

Accessory

Obesity, 225 pounds.

Arteriosclerosis of the cerebral arteries, moderate; and of the aorta and coronary arteries, advanced, with marked narrowing of anterior descending branch of the left coronary artery.

Sclerosis of the anterior leaflet of the mitral valve, moderate.

Recent and organizing hemorrhages beneath an arteriosclerotic plaque in the circumflex branch of the left coronary artery.

Hypertrophy and dilatation of the heart, 450 grams.

Foci of fibrosis in myocardium.

Fat infiltration of the myocardium, slight.

Accessory ostium of the right coronary artery.

Calcified subpleural nodule in the upper lobe of the right lung and caseocalcified tracheobronchial lymph node, right.

Small fibrous nodules in the parenchyma of the spleen.

Ascariasis of the intestine, 1 adult worm.

Diverticulosis of the sigmoid colon, slight.

Nodular hyperplasia of the prostate with median bar formation, slight.

Multiple retention cysts of both kidneys.

Accessory renal artery, slight.

Osteoarthritis of the spine, slight.

Osteoporosis of ribs, vertebrae, and sternum, slight.

Hallux valgus with prominence of the head of the first metatarsal of the left great toe.

Flexion deformity of the proximal interphalangeal joint of the right fifth finger.

Deformity of the bridge of the nose (history of broken nose).

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Is each physician conscious of the fact that good public relations for the medical profession begins in *his* office?

McIlvain Career Investigatorship

The Board of Directors of Community Studies, Inc., and the Department of Medicine of the University of Kansas School of Medicine announce the establishment of the McIlvain Career Investigatorship at the University of Kansas School of Medicine and the appointment of Dr. Santiago Grisolia as the McIlvain Career Investigator. The appointment became effective May 1.

A grant of $54,000 has been made to the school by Community Studies, Inc., from a trust established by the late Mrs. Frederic E. McIlvain in 1950. Dr. Sam Roberts, medical advisor to the trustees of the estate, is assisted by a committee composed of Doctors E. Grey Dimond, Kurt Reissmann, Stanley Morest, Blaine Hibbard, Harold Grady, and Robert E. Bolinger.

Dr. Grisolia was selected as the investigator on the basis of his research record and because his specific field—enzyme, hormone, and electrolyte changes in heart disease—seems to hold promise in the study of heart disease. He is a graduate of the University of Barcelona Faculty of Medicine and is coming to Kansas from the Enzyme Institute, University of Wisconsin, where he has been assistant professor of physiological chemistry. At the Kansas school he will be an associate professor of medicine and biochemistry and a member of the Department of Medicine and of the Cardiovascular Laboratory.

Successful Cancer Conference

The Sixth Annual Mid-West Cancer Conference, held April 1 and 2 at the Broadview Hotel, Wichita, was characterized by a record breaking attendance and a good, practical scientific program. Although most of those attending were from Kansas, physicians from neighboring states were also registered.

Dr. Karl E. Voldeng, Wellington, president of the American Cancer Society, Kansas Division, announced that the 1955 conference has been set for March 24 and 25. That session also will be held at the Broadview Hotel, Wichita. Dr. Homer L. Hiebert, Topeka, has been named as chairman of the committee to arrange the scientific program.

Only 100 years have passed since Kansas appeared on the map. It was in 1854 that the President of the United States, Franklin Pierce, signed an act establishing the Territory of Kansas. It was in 1859 that the governor of the Territory of Kansas approved the Act of Incorporation of the Kansas Medical Society.

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ACTIVITIES OF MEMBERS

Dr. Charles F. Haughey, who has been taking postgraduate work in surgery in Chicago and New Orleans, returned recently to his practice in Tribune. Dr. R. N. Todd, who cared for Dr. Haughey's practice, will go to Kansas City in June to begin a residency in radiology at the University of Kansas Medical Center.

Dr. H. M. Wiley, Garden City, spoke on cancer before the Dighton Rotary Club last month and also addressed an all-county cancer meeting there the same evening.

Dr. R. E. Riederer, Olathe, addressed the Central Parent-Teacher Association there recently on "A Medical Man Looks at Health and Accident Insurance."

Dr. George M. Gray, Kansas City, observed his 98th birthday anniversary in March. A story on the activities of the retired physician was carried in the *Kansas City Kansan.*

Dr. Edward Goldsich, who has practiced in St. Marys for four years, has announced plans to move to Kansas City, Missouri, for a residency at St. Luke's Hospital. Dr. John Montague, a graduate of St. Louis University School of Medicine who interned 18 months at Mercy Hospital, Denver, will take over Dr. Goldsich's practice.

Dr. Warren F. Bernstorf and Dr. Lynn C. Hartman took part in the Cowley County Health Education Workshop held at Winfield, March 18 and 19.

Dr. Eugene C. Kane, who has been associated with the Scott City Clinic during the past two years, has announced his resignation from the staff of the clinic, effective June 30.

Dr. David Robinson, of the University of Kansas Medical Center, presented a paper, "Prevention and Treatment of Scars," at a meeting of the Missouri State Medical Association held last month.

Dr. George W. Hammel, El Dorado, addressed the nurses' club in that city recently on the subject of "Cardiac Emergencies" and showed a film, "Wonder Engine of the Body."

Dr. Herbert R. Schmidt, Newton, returned recently from a trip to Paraguay where he visited his brother, Dr. J. R. Schmidt, and Mrs. Schmidt who are part of a Mennonite medical team working among lepers there.

Dr. C. V. Black, Pratt, addressed the grade school Parent-Teacher Association there recently on the subject of public health.

Dr. Raymond W. Lance, who practiced in Moline before entering service with the Air Force, was recently released from active duty and is opening an office in Arma.

The Lions Club in Girard recently heard a talk on "Community Health" given by Dr. Robert W. Friggeri.

Dr. Dick B. McKee, representing the Kansas State Board of Health, was one of the speakers at a youth health education conference held at Kansas State Teachers' College, Pittsburg, last month.

Dr. W. Clarke Wescoe, dean of the University of Kansas School of Medicine, was principal speaker at a health education tea given by the Woman's Auxiliary to the Wyandotte County Medical Society in Kansas City recently.

Dr. John L. Lattimore has been named to serve as chairman of the Legislative Committee of the Topeka Chamber of Commerce.

Dr. Richard L. Sutton, Jr., of the University of Kansas Medical Center, spoke on "Early Stages, Recognition, and Treatment of Cancer of the Skin" before the Alabama Division of the American Cancer Society at a meeting held recently in Birmingham.

At an area meeting of the American Cancer Society held in Salina recently, Dr. John H. Lathrop, Concordia, was elected medical representative from the 10th district to the state board. Dr. Donald D. Dieter,

REPORT ON Apresoline
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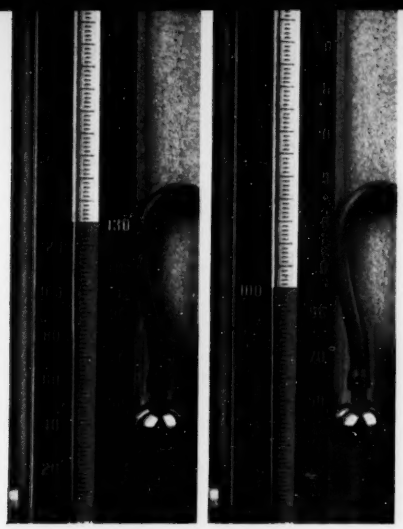

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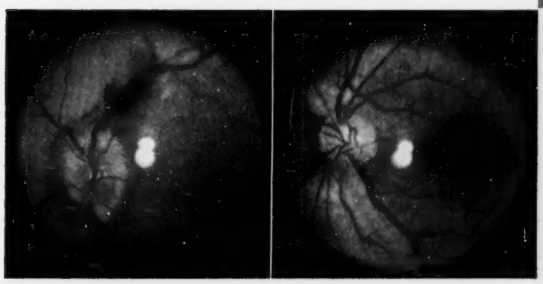

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1. TAYLOR, R. D., DUSTAN, H. P., CORCORAN, A. C., AND PAGE, I. H.: ARCH. INT. MED. 90:734 (DEC.) 1952.

*THE NORMAL FUNDUS (RIGHT) AS COMPARED WITH THE FUNDUS IN HYPERTENSION SHOWING EDEMA, EXUDATES, AND HEMORRHAGES (LEFT); ILLUSTRATIONS FROM "THE FUNDUS OF THE EYE": BEDELL, A. J.: CIBA CLINICAL SYMPOSIA 4:135 (JULY) 1952. THESE ILLUSTRATIONS ARE FOR DEMONSTRATION PURPOSES ONLY AND DO NOT REPRESENT APRESOLINE-TREATED PATIENTS.

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The above photos show a case of allergic rhinitis before and after Pyribenzamine therapy. Many such cases have been reported in the literature. A few examples: Loveless and Dworin[1] found Pyribenzamine beneficial in 82% of 107 patients; Feinberg[2] noted relief in 82% of 254 cases; Gay and associates[3] in 76% of 51 cases; Arbesman and colleagues[4] in 84% of 106 cases. In a later study Arbesman[5] rated Pyribenzamine one of "the most effective of all the drugs studied in allergic rhinitis. . . ." *Side effects:* It has been stated that "undesirable symptoms from the use of 50 to 100 mg. doses of Pyribenzamine were rarely of sufficient severity to interfere with its use."[6] Drowsiness, nausea, epigastric distress, vertigo and other side effects—rarely severe—may occur in some patients.

CONTROLS PENICILLIN REACTIONS

Pyribenzamine has been used successfully to control penicillin reactions—especially urticaria and itching. For example, Kesten[7] found that oral Pyribenzamine relieved or suppressed post-penicillin urticaria in 16 of 18 cases; she termed it "a most useful agent in allergic symptoms which follow the administration of antitoxin or penicillin."

RELIEVES ALLERGIC DERMATOSES

Foster[8] reported good results with oral Pyribenzamine in patients with various allergic dermatoses. In another study[9] of 241 such patients, Pyribenzamine was found effective.

C I B A

Pyribenzamine 25-mg. tablets now available— for children and for adults who can be maintained on low dosage or who experience side effects from the usual dosage of antihistamines

Supplied: Pyribenzamine hydrochloride 25-mg. and 50-mg. tablets; Pyribenzamine Elixir, 30 mg. Pyribenzamine citrate (equivalent to 20 mg. tripelennamine hydrochloride) per 4-ml. teaspoonful; Pyribenzamine hydrochloride solution (for parenteral use), 25 mg. per ml., in 1-ml. ampuls.

PUBLISHED CLINICAL STUDIES SHOW THOUSANDS OF ALLERGIC PATIENTS RELIEVED BY

Pyribenzamine®

PYRIBENZAMINE HYDROCHLORIDE (TRIPELENNAMINE HYDROCHLORIDE CIBA)
PYRIBENZAMINE CITRATE (TRIPELENNAMINE CITRATE CIBA)

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For complete information on Pyribenzamine ask your CIBA representative or write Medical Service Division, CIBA Pharmaceutical Products, Inc., Summit, N. J.

REPORT ON Pyribenzamine

EFFECTIVE ANTIHISTAMINE

PRODUCT OF CIBA RESEARCH

REPORT ON Priscoline

EFFECTIVE PERIPHERAL VASODILATOR

PRODUCT OF CIBA RESEARCH

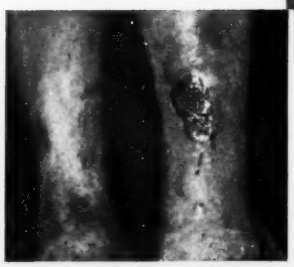
INCREASES PERIPHERAL BLOOD FLOW:

Priscoline reported to be a valuable aid to conventional therapy in peripheral ischemia and its sequelae—pain, loss of function, ulceration, gangrene, other trophic manifestations; Priscoline most effective when vasospasm is prominent but may prove limb-saving even when vasospasm is minimal because it decreases vascular tone, promotes establishment of collateral circulation.

MULTIPLE ACTION:

Priscoline exerts direct vasodilating effect on vessel wall, blocks sympathetic nerves (probably at their terminations in vascular muscle), blocks vasoconstrictive action of circulating epinephrine-like substances.

Side Effects: Certain side effects of Priscoline—"crawling" cutaneous sensation, chilliness with resultant gooseflesh or feeling of warmth—indicate attainment of effective dosage level; occasionally tachycardia, tingling, nausea and epigastric distress, slight hypotensive effect or slight rise in blood pressure may be experienced.



AGE 75. Arteriosclerotic ulceration with erysipeloid reaction and marked inflammation; after administration of oral Priscoline, 25 mg. three times daily, for one week—increased thereafter to 50 mg. four times daily—there is steady improvement, healing in eight weeks. No other medication used.

Priscoline®

FOR COMPLETE INFORMATION on Priscoline ask your CIBA representative or write Medical Service Division, CIBA Pharmaceutical Products, Inc., Summit, N. J. **SUPPLIED:** Priscoline hydrochloride (tolazoline hydrochloride CIBA) is available as 25-mg. tablets (scored), bottles of 100 and 1000; elixir, 25 mg. per 4 ml., in pints; 10-ml. multiple-dose vials, 25 mg. per ml.

Photographs and accompanying clinical data by courtesy of R. I. Lowenberg, M.D., Consultant in Vascular Surgery, Connecticut State Hospital, Middletown, Connecticut.

AGE 68. Arteriosclerosis obliterans cellulitis; sluggish response to saline dressings and procaine penicillin 300,000 units daily; healing speeded by oral Priscoline, 25 mg. four times daily for one week, 25 mg. every three hours thereafter; healing within six weeks.

3/7307

Salina, presented a film at the meeting, "Cancer—A Research Story," and conducted a question and answer session.

Dr. Lucien R. Pyle, Topeka, took part in a panel discussion on interprofessional relations during the annual meeting of the Kansas Pharmaceutical Association held in Topeka last month.

Dr. Karl E. Voldeng, Wellington, was speaker at a recent meeting of the Sumner County Medical Assistants' Society.

Dr. Frederick P. Wolff, who is in the service and is stationed at Camp Pendleton, California, was given a leave of absence in February to return to Pratt to aid Dr. M. E. Christmann who is recovering from injuries received in an automobile accident.

Dr. John P. White and Dr. Earl A. Martin, Parsons, were speakers at a three-day conference of nurses of the Southeast Kansas area held in Parsons in February.

Dr. Thomas L. Foster, Halstead, spoke on "Mental Hygiene" before a meeting of the Parent-Teacher Association in Canton in February.

Dr. DeWitt S. Lowe, Hiawatha, former medical missionary for the Presbyterian Church in South Korea, was the principal speaker at the Topeka observance of World Day of Prayer in March.

Dr. James A. Dugger, Topeka, recently became a diplomate of the American Board of Pediatrics.

Dr. Janet L. Holloway has returned to practice in Wichita after having completed a two-year residency in anesthesiology at the Lahey Clinic.

Dr. and Mrs. John L. Lattimore, Topeka, recently returned from a six weeks tour of Europe.

Dr. Robert M. Knox, who practiced in Wamego before going to Iowa Methodist Hospital in Des Moines for a residency in surgery, has announced plans to join the staff of the Nelson Clinic, Manhattan.

Dr. Joseph Gendel, Topeka Medical Center, has completed the examinations of the American Board of Orthopedics and is now a diplomate of the Board.

Dr. James G. Hughbanks, Dr. Albert E. Bair, and Dr. Vernon M. Lockard took part in a medical forum on the subject of cancer in Independence in March. A series of panel discussions on medical topics is being sponsored there by the Independence Rotary Club.

Dr. and Mrs. C. Omer West, Kansas City, left in February for a flight around the world.

Dr. N. C. Nash, Wichita, spoke on "Difficulties Encountered in Upper Gastrointestinal Tract Examinations" before a spring meeting of the Jackson County (Oklahoma) Medical Society in Altus.

Dr. Simon E. Hershorn, who has been serving in the Army, has received his discharge and has returned to practice in Wichita.

Dr. Lottie F. Law, Hill City, who has been practicing 52 years, announced her retirement recently. Two years ago, on the occasion of her golden anniversary in practice, 2,000 members of the community joined in a celebration in her honor. Her son, Dr. Findley Law, practices in Bird City.

Dr. John B. Dixon, Parsons, presented a paper on "Headaches" before a meeting of the Southeast Kansas Optometric Society on March 29.

Dr. J. D. Smith, who has been practicing in Hugoton during the past year, announced a change of location on April 1. He is now practicing in Satanta.

Dr. Edward A. Marrs, who has practiced in Sedan for 32 years, has retired and has moved to Colorado Springs.

Dr. Mervin T. Sudler and Dr. Monti L. Belot, Lawrence, announce that Dr. Glen A. Lessenden,

formerly of Tonganoxie, is now associated with them in practice.

Dr. E. Grey Dimond, of the University of Kansas Medical Center, is the author of a book, *Electrocardiography,* published last month by the C. V. Mosby Company, St. Louis.

Dr. Paul Lorhan, of the University of Kansas Medical Center, was speaker at a meeting of the Lincoln County, Nebraska, Medical Society recently. His subject was "Obstetrical Anesthesia."

Dr. Clarence H. Benage, Pittsburg, has been named by the Board of Trustees of the American Medical Association to the Commission on Improvement of Patient Care. The commission is made up of representatives of the A.M.A., the American Hospital Association, the American Nurses' Association, and the National League for Nursing.

Dr. Robert A. Jordan, Lawrence, has been appointed to the medical staff of the University of Kansas School of Medicine as assistant professor of medicine, director of the medical service of the outpatient department, and director of the student health center.

BLUE SHIELD

New Educational Program

Beginning in April, Blue Cross and Blue Shield Plans across the nation heralded the start of their first national program of public education.

A program of national magazine space has been outlined in three publications that will enable both Blue Cross and Blue Shield to explain their unique qualities in the field of prepayment health care.

Blue Cross began its advertising program with two-color, two-page spreads in LIFE, SATURDAY EVENING POST, and LOOK. Blue Shield is using the same media in different issues of the same magazines. Although Blue Cross and Blue Shield will have individual campaigns, there will be cross references in each advertisement to each other.

"The need for bringing the Blue Cross and Blue Shield stories to the American public in some continuing, co-ordinated way has become increasingly evident," Frank E. Smith, director of the Blue Shield Commission, points out. "Blue Cross and Blue Shield," he said, "were organized primarily as community services and not as commercial ventures. They are dedicated to serving the American people and fulfilling a social purpose. The new program of national education will attempt to point this out."

It is believed by the plans endorsing the national program that a strong conviction can be built in readers' minds to help them properly evaluate the welter of promises being made today in the hospital and medical protection market place.

Space in the national publications gives Blue Cross and Blue Shield the latitude necessary for their stories, as well as affording the plans the most effective and practical way to educate their present members and prospective members.

"Logically, if members really understand what their membership means, they will make a greater effort to retain it," Smith said. "At a cost far less than that offered by any other method, the new means of education will contact these members regularly in their homes. Wives, as well as husbands, will be exposed to the meaning of belonging to Blue Cross and Blue Shield."

Present plans call for nine Blue Cross messages in LIFE, eight in the POST, and eight in LOOK. The Blue Shield messages will appear five times in LIFE, five times in the POST, and four times in LOOK. Combined circulation of the three magazines for any one issue is 71 million.

Contributions from local Blue Cross and Blue Shield Plans are financing the national advertising program. Kansas Blue Cross-Blue Shield has not committed itself to participate at this time.

Dr. Howard Schriver, president of the Blue Shield Commission, has this to say about the national educational program: "We have felt for a long time that one barrier to even greater growth has been our lack of communication with the millions of people who want to know about Blue Shield yet are not exposed to the organization's story. When the first national Blue Shield advertisement appeared, we took the first major step toward telling our story on a national multimillion level."

COUNTY SOCIETIES

A meeting of the Tri-County Society was held at Harper in February. The program featured case reports by physicians from Sumner, Harper, and Barber counties.

Dr. Alfred Tocker, Wichita, was guest speaker at a recent meeting of the Barton County Society held at the Great Bend Country Club. His subject was

"Surgery of the Chest and Heart." Members of the Pawnee County Society were guests at the meeting.

Dr. David Culp, of the State University of Iowa College of Medicine, spoke on "Treatment of Carcinoma of the Prostate," before the Sedgwick County Society at its March meeting at the Lassen Hotel, Wichita. In April the group heard Dr. F. H. Ellis, of the Mayo Clinic, who discussed "Breast Surgery."

Dr. James J. Marchbanks, Oakley, was named president of the Northwest Kansas Society at a meeting held in Colby recently, and Dr. Edward E. Johnson, Norton, was chosen as vice-president. Dr. John S. Oney, Oakley, is secretary of the group.

Dr. W. J. Singleton, LaCrosse, was elected president of the Rush-Ness Society at a meeting held recently at the Rush County Memorial Hospital. Dr. J. H. Baker was named secretary, and Dr. F. K. Bowser will serve as delegate to the state meeting with Dr. Kenneth McLain as his alternate.

A combined meeting of the medical societies and the auxiliaries of the First District was held at Centralia in February. Guest speakers were Dr. Lucien R. Pyle, Topeka, president of the Kansas Medical Society, and Mrs. I. Joseph Waxse, Oswego, president of the auxiliary for the state. The meeting was arranged by Dr. W. L. Anderson, Atchison, and Mrs. C. M. Barnes, Seneca, councilors for the two groups.

"The Clinical Course of Poliomyelitis" was the subject of a paper given the Shawnee County Society by Dr. J. G. McQueen, Iowa City, at a meeting held in Topeka on March 1. At a meeting held on April 5 Dr. John Berry, Denver, spoke on "Cirrhosis of the Liver." Dr. Howard U. Kennedy was nominated as a trustee for Blue Cross-Blue Shield.

The Shawnee County Society has completed organization plans for civil defense. Members are assigned to one of three hospital units. Unit captains are Dr. Byron J. Ashley, Dr. D. R. Bedford, and Dr. George Hopkins. Assistant captains are Dr. Floyd C. Taggart, Dr. Charles E. Joss, and Dr. William Delaney. Dr. Dwight Lawson and Dr. Lucien R. Pyle are captain and assistant captain of a mobile support unit.

Five first aid teams have been assigned to different areas. Captains are Dr. Vernon M. Wiksten, Dr. A. J. Revell, Dr. L. L. Robbins, Dr. Clark Case, and Dr. Shirley Clark, and assistant captains are Dr. C. K. Schaffer, Dr. Evalyn Gendel, Dr. Seth Hammel, Dr. John Adams, and Dr. Lloyd Schwartz.

Members of the Harvey County Society were guests of the new Prairie View Hospital, Newton, at a dinner meeting held last month at the hospital. Additional guests were Dr. Thomas Morrow and Dr. F. Carter Newsom, Wichita, part-time staff psychiatrists at the hospital, and several Reno County physicians. The hospital, one of three mental institutions in this country operated by those of the Mennonite faith, contains 40 beds and will be open to persons of all creeds.

Dr. F. H. Ellis, of the Mayo Clinic, was guest speaker at a meeting of the Sedgwick County Society held at the Lassen Hotel, Wichita, on April 6. At the May meeting, on the 11th, members of the society presented a clinicopathological conference.

Members of the Johnson County Society discussed civil defense at a meeting held at the Milburn Country Club late in February. Dr. J. E. Schiffmacher, radiologist at St. Joseph Hospital, Kansas City, Missouri, spoke and presented a film depicting civil defense organization in Syracuse, New York. Dr. R. E. Riederer and Dr. G. J. Pierron were re-elected as president and secretary of the society.

A meeting of the Cowley County Society was held at the Arkansas City Country Club on March 18. Dr. Fred V. Hein, consultant of the Bureau of Health Education of the American Medical Association, spoke on "Physician Participation in the School Health Program." Members of the auxiliary were guests of the doctors at a dinner preceding the meeting.

A dinner meeting of the Nemaha County Society was held at Sabetha on April 8 with the two state legislators of that area as guests. Dr. Lucien R. Pyle, Topeka, led the discussion on legislative matters.

There are 220,104 physicians in the United States today, 156,433 of whom are in active practice, according to the A.M.A. On the basis of an estimated population of 160,000,000 in 1953, there is one physician actually engaged in the practice of medicine for every 1,000 persons.

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

These spring days are growing into weeks that really count in Congress. Unless a bill deals with an emergency, it had better be well on its way through committees by now or its chances of enactment will fade rapidly as summer approaches.

For good or evil, a large amount of health legislation is well advanced, and if Congress holds to an average pace several bills affecting the medical profession are likely to become law in the next month or so. Here is the situation in brief:

MEDICAL DEDUCTIONS. Legislation to increase the amount deducted from taxable income for medical expenses is a part of the omnibus tax revision bill which cleared the House early and by a wide margin, but ran into some delay on the Senate side. This bill, with the medical deduction liberalization intact, should reach the White House in plenty of time.

HILL-BURTON EXPANSION. A move to make important changes in this bill developed in the Senate Labor and Welfare Committee, after the House had passed its version with some amendments. American Hospital Association proposed that the rather complicated House legislation be scrapped, and instead that the Hill-Burton Act be amended to (a) include rehabilitation centers and nursing homes, and (b) place a high priority on hospitals for the chronically ill. The A.H.A. idea immediately attracted support in and out of the committee. The new approach meant inevitable, but probably not fatal, delays.

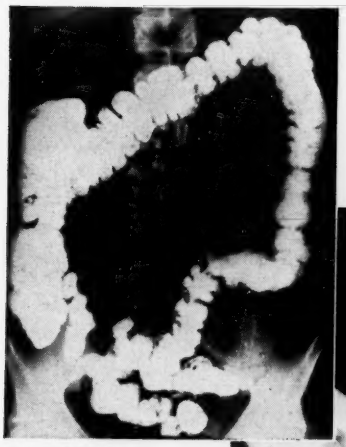
REINSURANCE. This proposal, once hailed as the keystone of the Eisenhower administration's health program, continued to encounter opposition. At one stage, of all the national associations to testify on reinsurance only American Hospital Association was giving it unqualified support. American Medical Association, the U. S. Chamber of Commerce, and national spokesmen for the insurance industry took about the same position: (1) Reinsurance alone cannot make uninsurable risks insurable. (2) The threat of federal control of medicine is inherent in any program that would bring the federal government in such close contact with medical practice. Dr. David B. Allman, representing the A.M.A. at the House hearings, emphasized that the association would welcome and co-operate in any movement carrying real promise of promoting voluntary health insurance.

HEALTH GRANTS. This is an administration plan to do away with the present categorical grants for identified projects, such as venereal disease control, and to substitute funds earmarked for three general purposes: (a) to maintain present programs, (b) to initiate new programs or to expand existing ones, and (c) to finance public or private experimental or pilot programs of national or regional significance. In both committees the question was whether to group the first and second type grants together, with the state health authorities deciding how to divide up the federal money among old and new projects. Funds for the third type grant—experimental—would be completely controlled by the surgeon general. One suggestion is to require approval of the state health officer for any experimental (type three) grant in his state. Another is to eliminate the third type grants altogether, letting the National Institutes of Health handle public health as well as other medical research grants.

SOCIAL SECURITY. American Medical Association, American Dental Association and a number of other national groups are fighting vigorously to prevent compulsory extension of Old Age and Survivors Insurance to physicians, dentists, and most other self-employed. Instead, they want the privilege of deferring income tax payments on that part of earnings placed in restricted annuities—the Jenkins-Keogh plan. A.M.A. also feels that there is no need for the bill's provision that pension rights be frozen during periods when the worker has been medically determined to be disabled. A better suggestion, the Association maintains, is to base pension rates on the 10 best working years, thus virtually eliminating the need for the controversial medical examinations. Prospects are good that social security will be extended, either with or without these changes.

VOCATIONAL REHABILITATION. Generally, Senate witnesses favor the administration's proposal to expand the federal-state programs, providing U.S. grants aren't cut. However, with no House bill introduced as of this writing, there is some doubt that, even if the Senate clears the measure, the House can find time to deal with it.

DOCTOR DRAFT AMENDMENT. This bill, an outgrowth of the Peress case, swept through the Senate without objection. It may be law by the time this is published. It would amend the Doctor Draft act to permit the services to keep on duty as an enlisted man, assigned to professional tasks, anyone called under the Doctor Draft act whose loyalty is questioned. Defense Department has promised to investigate such cases immediately, so that the man can be cleared promptly and offered a commission or discharged. The discharge would state that action was taken on loyalty grounds.



Normal Colon

Ulcerative Colitis

Atonic Colon

Smoothage and Bulk in Correcting Constipation

To initiate the normal defecation reflex, the "smoothage" and bulk of Metamucil® provide the needed gentle rectal distention.

Once the habit of constipation has been established, due to any of a large number of causes, it becomes a major problem. Self-medication with irritant or chemical laxatives, or repeated enemas, usually causes a decreased, sluggish defecation reflex and may result in its complete loss.

Rectal distention is a vital factor in initiating the normal defecation reflex, and sufficient bulk is thus of obvious importance in restoring this reflex. Metamucil provides this bulk in the form of a smooth, nonirritating, soft, hydrophilic colloid which gently distends the rectum and initiates the desire to evacuate. Metamucil demands extra fluid, imparting even greater smoothage to the intestinal contents.

It is indicated in chronic constipation of various types—including distal colon stasis of the "irritable colon" syndrome, the atonic colon following abdominal operations, repressions of defecation after anorectal surgery and in special conditions such as the management of a permanent ileostomy. Metamucil is the highly refined mucilloid of Plantago ovata (50%), a seed of the psyllium group, combined with dextrose (50%) as a dispersing agent.

The average adult dose is one rounded teaspoonful of Metamucil powder in a glass of cool water, milk or fruit juice, followed by an additional glass of fluid if indicated.

Metamucil is supplied in containers of 4, 8 and 16 ounces. It is accepted by the Council on Pharmacy and Chemistry of the American Medical Association. G. D. Searle & Co., Research in the Service of Medicine.

Treatment of Hepatic Insufficiency

Helen Jones, M.D.

Tucson, Arizona

"He who would know correctly before hand those that will recover and those that will die, and in what cases the disease will be protracted for many days and in what cases for a shorter time, must be able to form a judgment from having made himself acquainted with all the symptoms and estimating their powers in comparison with one another." Hippocrates' *Book of Prognostics.*

A consideration of the immediate effects of injury upon composite organs reveals that, in general, those cells which are most highly differentiated, and which endow the organ with its characteristic function, are also those which are most susceptible to damage. Nowhere is this more evident than in the liver.[1]

The term "hepatic insufficiency" may be defined according to the usage of the individual observer of hepatic disease. There would probably be a uniform acceptance of the term for the comatose or disoriented patient with a severe hepatic disorder, such as diffuse necrosis or cirrhosis. It might also be used to apply to more serious clinical states associated with diffuse parenchymal hepatic disease, with jaundice and ascites, but without obvious mental depression or aberration. It would seem helpful to define "hepatic insufficiency" as that state in which the patient's normal economy and activity is seriously interfered with because of a diffuse hepatic injury; in other words, a state in which the functions of the liver are unable to meet the general needs of the individual.[2]

Cirrhosis of the liver with ascites and massive hemorrhage presents a well known clinical picture, but the less severe forms of chronic liver disease are usually not diagnosed. Yet it is probable that various types of low-grade chronic liver disease are quite common. Our failure to recognize these cases clinically is apparently due to an absence of a clearly defined and well established clinical picture. Symptoms and physical findings are not striking, and definitive laboratory procedures are often lacking. However, it does not seem reasonable to assume that disease of an organ which plays such a varied and important role in the body economy as the liver is entirely "silent" and without clinical manifestations.[3]

Consideration of the clinical types of liver disease fully supports the conclusion derived from a study of its pathology, that the same type of liver injury may, on occasion, be caused by several different factors. The clinical manifestations of liver injury from widely different causes may be indistinguishable, and it is only by attention to associated general effects of the causative agents or exposure to special conditions that the exciting cause of the illness can be recognized. Only in the broadest sense can the clinical manifestations of liver injury be related to the particular pathological lesion present.[1]

The earliest lesion in massive hepatic necrosis shows gross congestion of the portal tracts, periportal hemorrhage, and swollen dead parenchymal cells throughout the lobule. It seems that in this condition the parenchyma has swollen to such an extent as to arrest completely the intralobular circulation. Death of every parenchymal cell throughout the lobule—massive necrosis—is inevitable. A similar impeding of the intralobular circulation is produced by fatty infiltration. This is sufficient only to lead to quiet degeneration of the parenchyma.[4]

Necrosis of the liver is followed by fibrosis, of which there seem to be two distinct types. Post-necrotic scarring, which usually follows the massive type of necrosis, leaves an organ cut into by coarse bands of fibrous tissue with separate areas composed of normal liver lobules. The other is the diffuse fibrosis which begins as a thickening of the reticulin fibers in the region of the portal tract or central veins and involves every lobule. It is clear that the anatomical form of the original hepatic lesion has a bearing on the consequences of hepatic injury, and it would seem also to have an important influence on survival in patients with liver disease.[5]

From the evidence now available, it is believed that the prognosis in cases of so-called "alcoholic" cirrhosis is related to the degree of portal fibrosis, bile duct proliferation, and adenoma formation with resultant shrinking and distortion of normal liver parenchyma. These changes roughly indicate the extent of previous destruction of liver cells. In other words, the reversibility of hepatic insufficiency would appear to depend upon the remaining amount of liver parenchyma not damaged beyond repair. There is little doubt that a fatty cirrhosis, in which the enlargement is due mainly to fat rather than to adenomata and connective tissue, offers a much better prognosis than one of the latter type, even though it is still enlarged.[2]

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school.

Thesis written while the author was a fourth year student. Dr. Jones is now interning at St. Mary's Hospital and Sanatorium, Tucson, Arizona.

Meats-in-a-Can

and Kitchen-Cooked Meats... Comparative Nutritive Values

From a practical dietary standpoint, meats-in-a-can—preserved by commercial canning—are nutritionally interchangeable with meats of like variety prepared in the home.[1] For taste appeal, for economy and "keeping" quality, and for household convenience, meats-in-a-can are advantageous in many respects.

As the comparative data here shown indicate, kitchen-prepared meats and similar meats-in-a-can are closely alike in the amounts of various nutrients they provide.

COMPARATIVE COMPOSITION OF KITCHEN-COOKED AND COMMERCIAL-CANNED MEATS
(Nutrient Amounts per 100 Grams)

	*Kitchen-Cooked Ham[2]	**Canned Ham[3] (Chopped, Cured)	Kitchen-Cooked Beef Round[2]	Canned Roast Beef[2]
Water	50%	50%	59%	60%
Protein	21 Gm.	20 Gm.	27 Gm.	25 Gm.
Fat (ether extract)	28 Gm.	20 Gm.	13 Gm.	13 Gm.
Niacin	4.0 mg.	4.3 mg.	5.5 mg.	4.2 mg.
Riboflavin	0.21 mg.	0.19 mg.	0.22 mg.	0.23 mg.
Thiamine	0.46 mg.	0.40 mg.	0.08 mg.	0.02 mg.

*Values after conversion from 42% to 50% water basis.
**Values after conversion from 58.69% to 50% water basis.

Experimental studies have shown that the processing which meats-in-a-can undergo leads to little if any greater vitamin losses than does home-cooking of similar cuts of meat. In general, meats-in-a-can retain of their original vitamin content approximately:

60 to 80 per cent of thiamine
90 to 100 per cent of riboflavin
90 to 100 per cent of niacin
80 per cent of biotin
70 to 80 per cent of pantothenic acid.[4,5]

During storage for customary periods, at usual warehouse temperatures, meats-in-a-can show little, if any, further vitamin loss except in thiamine. Even thiamine, a highly thermolabile vitamin, was 52 per cent retained in pork-in-a-can after ten months' storage at 80° F. Retention of the vitamin was notably greater when the canned pork was stored at 38° F.

Since meats-in-a-can are thoroughly cooked in processing, they may be consumed as purchased, merely warmed or mildly cooked. When the meat is moderately cooked in preparation for consumption, little or no further loss in vitamins need to occur.

Recent studies show that meats-in-a-can are excellent sources of needed amino acids.[6] The 18 amino acids determined in these studies appeared in similar ratio and amounts in canned beef, pork, and lamb as in the respective fresh or home-cooked meats.

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The Seal of Acceptance denotes that the nutritional statements made in this advertisement are acceptable to the Council on Foods and Nutrition of the American Medical Association.

ACCEPTED COUNCIL ON FOODS AND NUTRITION AMERICAN MEDICAL ASSOCIATION

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Symptomatology is the expression of deranged function and, although gross alterations in structure lead to such derangements, disturbances of function may occur from lesions too subtle to cause structural alterations. By analyzing the symptomatology of established pathological lesions, it is possible to see its broad correlation with damage to the different structures in the liver, and thence to infer the site of disturbance in those illnesses in which pathological manifestations are less pronounced. The next contribution to the interpretation of symptomatology will undoubtedly come from a direct study of the gross functional disturbances themselves.[1]

Before any attempt of analysis of symptomatology may be made, however, one must first bear well in mind what symptoms occur in hepatic insufficiency. "There is commonly a manifestation which has been given too little attention and which indeed has been sniffed at too much, and sniffed of too little. This is the 'fetor hepaticus,' the perception of which often enables one to recognize hepatic insufficiency, even though jaundice may be slight or absent."[2]

Other signs often associated with, but not necessarily indicative of, hepatic insufficiency include palmar erythema, multiple spider-nevi, recent loss of axillary hair, gynecomastia, venous collateral circulation, and hemorrhagic tendency. Ascites and edema are common signs. The size of the liver is often of the utmost significance in prognosis. Rapid diminution is well recognized as one of the most ominous signs of all, indicating diffuse necrosis, and as a rule a fatal outcome, although even here exceptions are noted.[2] Symptomatology includes anorexia, nausea and vomiting, abdominal pain, diarrhea, and easy fatigue. A keen watch should be kept for the slightest sign of undue somnolence, disorientation, or euphoria, since these are often the first and ominous harbingers of hepatic coma.[2]

As has already been seen, there is considerable variance of opinion concerning the relationship between histology, symptomatology, and prognosis. Waldstein et al.[6] have attempted to correlate the major clinical and biochemical findings with the histologic features. These findings, in the majority of cases, may help the clinician predict the pathologic process (except possibly fatty metamorphosis) that is present in the liver and thus aid in the estimation of the stage and activity of the cirrhotic lesion. In keeping with the observations of others, the results indicate that the histologic findings at any one time have little long term prognostic significance. For the time being, prognosis is best judged on the clinical grounds that have been established.

Laboratory Tests: Hanger, of Columbia University, gives the following discussion of laboratory tests and their significance in prognosis of liver disease.[7]

More than 500 functions have been ascribed to the liver. Many of these represent complex enzymatic activities in the intermediary metabolism of foodsstuffs and are still beyond the range of clinical study. Others can be measured relatively easily in the human being and thus may be utilized to evaluate the status of hepatic efficiency in health and disease.

Histologic Features[6]	*I Liver Cell Damage*	*II Inflammation*	*III Scarring*	*IV Fat*
Clinical Findings				
Jaundice	x	..	..	..
High jaundice	x	..	..	x
Anemia	x	..	..	..
Ascites	..	x	x	..
Hyperestrogenism	..	..	x	..
Alcoholism	..	..	x	x
Biochemical Findings				
Flocculations	x	x	..	..
Globulins	x	x	..	..
Albumin-globulin ratio	x	x	..	..
Albumin	x	..	x	..
Total protein	..	..	x	..

The liver may show a wide variety of derangements which may be grouped with certain overlapping as follows: (1) impairment of one or many metabolic functions, (2) abnormalities in the excretion of various constituents of the bile, (3) disturbances of flow of blood through the liver or inadequate distribution of blood to hepatic cells, and (4) inflammation or scarring of the supporting structures of the liver.

It is not unusual to encounter derangements in all these categories during the course of diseases with widespread involvement. On the other hand, lesions of the liver may be highly selective, and, unless the appropriate type of test is employed, the disorder may be completely unrecognized.

Liver function tests are indicated under a variety of clinical circumstances: (1) for the diagnosis of an obvious hepatic derangement such as jaundice or hepatomegaly, (2) to follow the course of a disorder, or (3) for the detection of possible hepatic involvement among subjects who have been exposed to certain infections, noxious agents, or other hazards known to be debilitating to the liver.

In our clinic the following procedures for hepatic investigation are used almost exclusively:

1. For the detection of decreased metabolic activity: (a) total serum cholesterol with the determination of the esterified fraction, (b) serum albumin level, and (c) bromsulfalein clearance.

2. For the recognition of excretory difficulties: (a) total serum bilirubin with the determination of the prompt direct reacting fraction and (b) serum alkaline phosphatase.

3. For the recognition of necrotizing or inflammatory reactions in the liver: (a) cephalin flocculation, (b) thymol turbidity, and (c) serum globulin level.

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The various patterns of hepatic functional derangement encountered in hepatic disease are determined largely by the acuteness of cellular damage and the extent of involvement. Acute necrotizing lesions usually cause the prompt appearance of a positive cephalin flocculation reaction. The thymol turbidity reaction tends to become positive as inflammatory reactions develop. Both of these tests, therefore, are indicated in the diagnosis of conditions such as acute and chronic hepatitis, active Laennec's cirrhosis, acute anoxia, and cases of poisoning with such agents as phosphorus, carbon tetrachloride, and other noxious agents.

Diets deficient in protein, chronic biliary obstruction, intrahepatic compression, chronic anoxia, repeated exposure to small doses of hepatotoxic agents, alcoholism, etc., lead to gradual damage with diverse manifestations, such as fatty infiltration, hyaline degeneration, atrophic changes, sluggish functioning, and depressed viability of parenchymal cells. Chronic injury often gives rise to selective impairment of hepatic functions, such as lowering of serum albumin, decreased esterified cholesterol fraction in the serum, impaired bromsulfalein excretion, etc., while other functions may be well maintained.

Post and Patek were able to correlate the prognosis of patients treated with a highly nutritious diet with the plasma serum albumin level at the time of the patient's admission to the hospital. On the average, those patients who died had a serum albumin of 2.4 grams per 100 cc. on admission, whereas those who improved and whose ascites disappeared had a serum albumin averaging 3.0 grams on admission, a difference which was statistically significant.[8]

It is obvious that flocculation or turbidity tests, which are usually negative or irregularly positive during the course of sluggish degeneration of hepatic cells, are of little value in the recognition of conditions such as fatty infiltration, chronic passive congestion, neoplastic invasion, and chronic poisoning, unless necrotizing or inflammatory lesions are also present. Further important information may be gained by repeating a small battery of pertinent tests at weekly intervals until the trend of the disease under standardized conditions has been established.

DIETARY FACTORS

The dietetic regimen of patients under Patek's care included approximately 350 grams of carbohydrates, 140 grams of protein, and 175 grams of fat, a total of 3,500 calories a day. The diet consisted chiefly of meat, fish or poultry, milk, eggs, fruit, and green vegetables. The diet was supplemented with a total of 50 grams of powdered brewer's yeast a day, or, if this was not tolerated, an oral B complex preparation. In addition, intramuscular injections of thiamine chloride, 5 mgm. daily, and unconcentrated liver extract, 5 cc. twice weekly, were given.[8, 9]

The intensive combined method of therapy for cirrhosis of the liver used by Morrison and his workers consisted of administering a maximal protein, low fat, high carbohydrate diet in which protein was forced to maximum tolerance by three meat servings and at least six to eight glasses of skim milk and cottage cheese daily. This basic diet was supplemented by daily injections of 5 cc. of whole liver extract fortified with high potency vitamin B complex in large dosage, by oral vitamin B complex and multivitamin capsules, and by two grams each of methionine and choline daily. Such a method resulted in a sharp reduction of mortality, morbidity, and disability rate in the series of patients with chronic hepatitis.[10]

It should be pointed out that the improvement in the prognosis of patients with cirrhosis under the care of Patek and his contemporaries occurred during the era of chemotherapy for infection, and this may account for some of the improvement noted.[8]

The ease with which patients can be induced to eat both the diet and supplementary feedings to a total of 3,500 to 4,000 calories per day is in itself one of the best indices of prognosis. Rapid improvement may be expected in those who will do this with but little urging, and in general the prognosis varies roughly with the appetite, being worse in those with severe anorexia, and, of course, worst in those who are comatose and will eat nothing.

Of crucial importance in all cases with anorexia is the attention given to the total food intake from day to day. All too frequently a sense of false security is gained merely by the fact that an adequate diet has been ordered or that certain amounts of glucose are being given intravenously, without due inquiry as to whether the 24-hour caloric intake has reached a desirable level. It is best that this be maintained on a current basis, from hour to hour. This requires a general awareness of the importance of the problem on the part of the attending physician, house officers, nurses, dietitians, and even the close members of the family, if intelligent and cooperative.[2]

In the work of Eckhardt et al.,[11] three deeply jaundiced patients with active fatty alcoholic cirrhosis were selected for a study of the effect of protein starvation for a short period of time, and of subsequent re-feeding upon their clinical course, liver function, and liver histochemistry. Improvement in clinical status and in liver function occurred in spite of negative nitrogen balance occasioned by diets grossly deficient in or devoid of protein. Liver histology, however, failed to reflect this improvement, and, except for a moderate reduction in liver fat during choline administration, morphologic improvement

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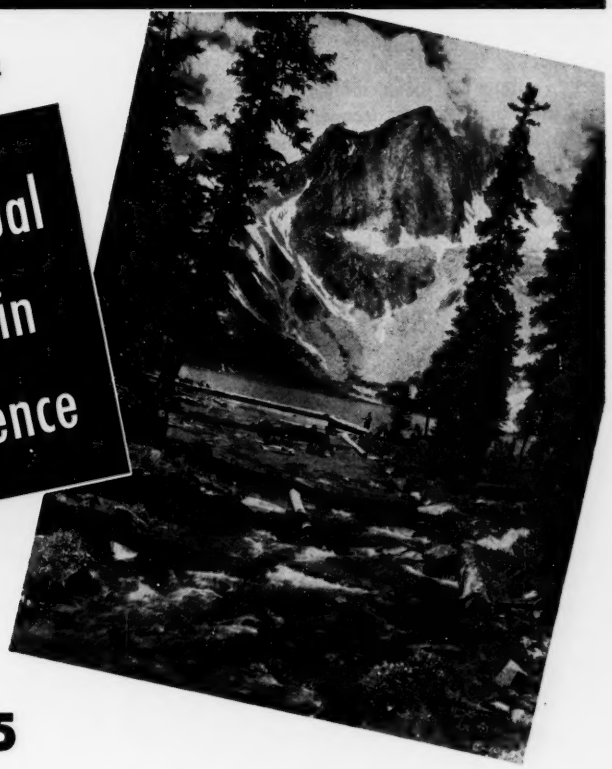

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appeared only with the provision of adequate exogenous protein and the maintenance of positive nitrogen balance.

It may be possible, however, to improve further the current regimen of treatment of chronic hepatitis by reducing the work now required of the liver. Energy is required to metabolize amino acids and to form urea from the nitrogen of the molecules. Possibly a decrease in the current high protein intake of 140 to 150 gm. per day might be feasible without a loss in efficacy of the treatment regimen, if lipotropic substances are furnished to the body.[12]

Necrosis and cirrhosis do not respond to the same dietary factors. Once the liver cells are destroyed through the stages of degeneration and necrosis, therapy may only hasten regeneration of new hepatic parenchyma, but it will not achieve direct repair of dead cells. Clinical management should therefore concentrate on the prevention of such serious complications as necrosis, or, if it is already present, on its containment. This may be attempted by a diet high in sulfur-containing amino acids, methionine and cystine, and in tocopherol.[13]

The liver is a junction in the body's transportation system for fat. The causes of the deposition of excess amounts of fat in the liver can be divided into three general groups: first, the mobilization of an abnormal amount of fat to the liver; second, a decrease in the effectiveness of the factors which normally promote movement of fat away from the liver; and third, interference with the normal ability of the liver cells to metabolize fat. It is obvious that under many circumstances more than one of these processes may be operative.[14] If hepatic enlargement is due to fatty metamorphosis or fatty cirrhosis, the prognosis is relatively good; it has become increasingly clear within the recent past that a good diet alone will frequently serve to induce a remission.[2]

Best and his group classify fatty livers on the basis of four general causes: (a) dietary abnormalities, (b) hormonal imbalance, e.g., insulin deficiency, (c) oxygen lack, e.g., anemia or ischemia of liver cells and, (d) toxic or infectious processes. Each type may respond to therapy designed to correct the specific etiological abnormality; there is no single treatment for all fatty livers.[15]

In cirrhosis and diffuse hepatic injuries which are primarily non-fatty and non-dietary in genesis, it is doubtful that dietary therapy is of nearly as great significance in prognosis or treatment. In fact, the more one studies this group, the more one is inclined to the belief that the physician's role is that of protecting the patient against further insult such as may arise from exertion, fatigue, malnutrition, negative nitrogen balance, drugs or chemicals, anesthesia, or needless cholecystectomy or exploration of the common bile duct.[2]

LIPOTROPIC FACTORS

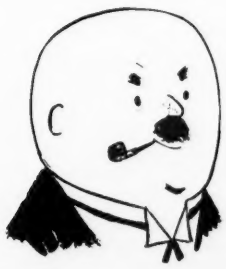
The liver can be made fatty by the addition of an excess of fat to a normal diet. This is, in part, due to a relative deficiency of choline. It can be made extremely fatty by withdrawal of the lipotropic agents from a diet, even if this contains no fat.[14] Fatty infiltration of the liver occurs when the neutral fats cannot be removed from the liver by such transport systems as the phospholipids. The liver is infiltrated with fat whenever the synthesis of phospholipids is impaired. This occurs when there is an insufficiency of essential fatty acids, when there is an insufficiency of choline and methionine, when excesses of cystine cause the utilization of methionine for the formation of protein, and so forth.[8]

The Spanish group has presented interesting data which suggest that the fundamental defect in phospholipid synthesis in hepatic disease is an enzymatic one and not a deficiency of building blocks, since not only was added choline lost almost quantitatively, but also in the presence of hepatic disease a spontaneous cholinuria was present, which subsided with clinical recovery.[16]

In human beings with liver disease, the therapeutic value of choline and methionine has not been definitely established. Choline or methionine is indicated only at the beginning of treatment in patients with fatty infiltration of the liver who are acutely ill and cannot eat. The same lipotropic effect is achieved more slowly without choline or methionine in patients who will eat an adequate diet.[17] While it seems true experimentally that choline exerts a beneficial effect by clearing the fat from the liver, therapeutic trials have not always yielded unequivocal results. The possibility of toxic effects must be taken into consideration. Particularly, the possibility of circulating acetylcholine appears of definite importance.[18]

Hepatotoxic substances such as chloroform, carbontetrachloride, and phosphorus can produce, depending on dosage and duration of administration, fatty changes, necrosis, and fibrosis of the liver. Dietary changes affect the toxicity of such agents. Liver injury may also be increased, probably due to the greater solubility of many of these substances in fat, allowing them a more prolonged toxic action of the compound. Depending on the hepatotoxic agent being used, its dose and route of administration, and the dietary conditions of the study, mortality may be influenced by choline, methionine, cystine, other compounds supplying SH groups, or totally unrelated agents. One would expect supplements of choline to act chiefly with regard to the prevention or alleviation of fatty changes and methionine and cystine to affect the incidence of necrosis.[19]

Further suggestions have been made concerning the application of lipotropic substance therapy. Since



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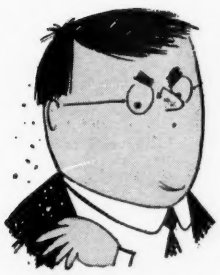

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stress, such as anesthesia, trauma, or infection, will induce a decrease in liver function in normal persons, one wonders whether it will prove possible to prevent liver damage by prophylactic administration of lipotropic and other substances when stress can be anticipated.[12]

VITAMINS

Parenteral administration of vitamins totaling several times the daily requirements is advisable.[20] Vitamin K preparations are clearly indicated in the presence of hypoprothrombinemia because of the possibility that defective alimentation is responsible. If reversion to normal with injection of small doses (2 to 4 mg. of the parenteral preparation) does not occur, large doses will probably be ineffective and may possibly be harmful. Thiamine in moderate doses, to aid in metabolism of the added glucose, is clearly indicated.[16] It has been shown that the pyruvic acid level in the blood and spinal fluid rises in direct proportion to the degree of liver failure and that the administration of thiamine does not check this rise.[20]

There is doubt that large quantities of purified vitamins are needed, despite the fact that the liver stores water and fat soluble vitamins. The defect again is probably one of faulty utilization. Niacin in massive doses may be injurious, since it is antilipotropic when excreted, using methyl groups which might otherwise be available for transmethylation or phospholipid synthesis.[16]

Recent work by György with experimental animals indicates that vitamin B_{12} may have strong lipotropic activity in fatty livers.[20]

INTRAVENOUS GLUCOSE

It is believed of value to administer glucose intravenously in any case of hepatic insufficiency in which anorexia is at all prominent, although the mere giving of intravenous glucose should not create the illusion that everything necessary is being done. Should insulin be given with the glucose? Proof of its virtue in hepatic insufficiency has not been provided; nevertheless, it may be beneficial at least in promoting appetite in certain cases.[2]

It should be pointed out, according to Karl et al. in their work with hepatic coma, that several patients who had received large quantities of glucose in water showed a marked drop in blood electrolytes. They had not had paracenteses or mercurial diuretics to account for low salt and potassium levels and must be considered to have had electrolyte dilution on the basis of water intoxication.[20]

It may be inferred that hepatic coma represents a functional disturbance rather than an anatomical one. The "lesion" may be a biochemical disturbance involving carbohydrate metabolism and which perhaps is incidental to a breakdown of certain enzyme systems (cozymase and cocarboxylase), which play a part in the intermediary metabolism of glucose. If these enzyme systems can be sustained and glucose supplied for combusion, there is a possibility of recovery, provided always that sufficient hepatic tissue remains to sustain life.[21]

PARENTERAL PROTEINS

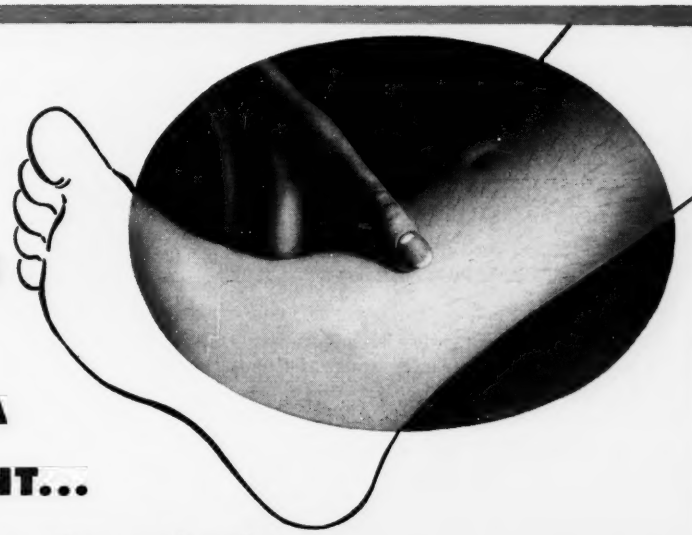
The question often arises whether the patient in coma from acute infectious hepatitis or cirrhosis should be given intravenous amino acids. On theoretical grounds this might be considered dangerous since the sick liver might not be able to metabolize the excess load of amino acids. Actually, however, no deleterious effects have been observed from the intravenous use of commercially prepared amino acids in patients in hepatic coma or ill with severe hepatitis or cirrhosis, although at the same time one would hesitate to ascribe any benefit to them.[8] Karl and his group state that comatose patients with severe liver damage should definitely be supplied proteins parenterally.[20]

Concentrated salt-poor human serum albumin is neither specific for the relief of ascites nor markedly beneficial in the treatment of the underlying liver disease. In view of the variety of results obtained with concentrated human serum albumin in patients with cirrhosis of the liver, it is concluded that other factors in addition to a reduced serum osmotic pressure, as represented by decreased serum albumin concentrations, are active in the retention of fluid.[22] Therefore, it seems reasonably conclusive that serum albumin is of little or no value for producing sustained diuresis, reducing ascites, or improving liver function, and that it may even induce fatal hemorrhage or pulmonary edema.[20]

LIVER EXTRACT

The use of intravenous liver extracts has been recommended for patients with Laennec's cirrhosis or hepatic insufficiency from other causes. Liver extracts were advocated for the treatment of liver disease as early as 1626. Numerous enthusiastic reports of their use appeared in the French literature early in the present century.[8] It is believed that one of the effects of crude liver extract administered in large doses intravenously is to restore and sustain appetite in previously anorexic patients with cirrhosis. Used in conjunction with a full diet, a great nutritional advantage is gained which results in the control of wasting and the restoration of protein storage.[23]

Ralli and her associates, in their work with the administration of large doses of crude liver extract, found the survival in the group treated with liver extract to be significantly more prolonged than in the patients treated in the conventional manner. She states that the fact that the patients receiving liver extract



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1. Abramson, Julius, Bresnick, Elliott, and Sapienza, P. L.: *New England Jour. Med.*, 243:44, July 13, 1950.

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survived longer and were in a better state of nutrition than the patients in the control group suggests that the growth-promoting factors in the liver extract may have been a factor in stimulating regeneration of liver cells. Patek's group, in whom the survival is better than that reported by other investigators, received dried brewer's yeast, which also contains growth-promoting factors.[5] Whatever its action, there is little question of its value in the treatment of liver disease.

ELECTROLYTE AND WATER BALANCE

Numerous agents have been employed in an attempt to control the ascites of hepatic insufficiency, including restriction of sodium intake, mercurial diuretics, and, more recently, the cation-exchange resins. Paracentesis, although often necessary as a last resort, is recognized by all to be an admission of inability to control water balance by any other means. Lowe and Overy state that since rigid dietary sodium restriction spares the patient with hepatic cirrhosis and ascites serious protein loss and the discomfort of frequent abdominal paracentesis, it is considered worthwhile. It is not without hazard, however. Close clinical surveillance is necessary to guard against the serious complication of sodium depletion. If sodium depletion develops, electrolytes should be replaced even if this results in the formation of ascites.[24]

One group reports that seven out of eight patients with hepatic cirrhosis had diuresis with diminishing peripheral edema and ascites following the use of an intravenously administered mercurial diuretic and salt-poor human albumin.[25] Hilton, on the other hand, finds that data indicate that in cirrhotic patients with ascites the renal excretion of potassium following the administration of mercuhydrin may, under certain circumstances, be of such magnitude as to produce hypokalemia and a clinical state indistinguishable in symptomatology from the previously described low salt syndrome. These patients were further characterized by their almost total inability to excrete dietary sodium chloride and their lack of response to mercurial diuretics.[26]

A syndrome indistinguishable from impending hepatic coma has recently been described in patients with cirrhosis of the liver given certain ammonium-containing cation-exchange resins. Elevated blood ammonia levels and an intolerance to certain nitrogenous substances in patients with cirrhosis of the liver suggested that this reaction was related to the ammonium contained in the resin. Untoward effects apparently identical with spontaneously occurring hepatic coma can be provoked in patients with cirrhosis of the liver by the administration of ammonium chloride, diammonium citrate, urea or dietary protein.[27] Further work is required to evaluate the cation-exchange resins fully.

ACTH

The use of ACTH in the treatment of liver disease is still in the experimental stage. Theoretically it should be of value in slowing down the process of fibrosis in the liver. It also seems to be of value in any severe toxic state, though the mechanism is unknown. Colbert et al. found that 5 patients with acute viral hepatitis who received 100 mg. of ACTH daily for periods ranging between 9 and 21 days, showed marked symptomatic improvement promptly. This was associated in all instances with a falling serum-bilirubin concentration. Untoward effects included arthralgia on withdrawal of ACTH, glycosuria, hypertension, edema, ascites, "moon face," and acne.[28]

Of interest in considering the action of ACTH is the work showing that a fat-mobilizing factor appears in the urine of fasting rabbits but is absent when the animals are fed. Since it has been shown that both the growth factor of the pituitary and ACTH exert this fat-mobilizing effect, it will be interesting to determine the nature and physiological actions of the material excreted by fasted animals, and thus learn whether it is an adrenal product or one of the pituitary factors.[14] Further time is needed for the collection of clinical results in the use of ACTH in the treatment of liver disease.

SURGICAL PROCEDURES

In treatment of cirrhosis of the liver, the hepatic artery may be ligated immediately distal or proximal to the gastroduodenal artery without producing necrosis of the liver. Similarly, the splenic artery may be ligated without producing necrosis of the spleen. However, the efficacy of the ligation of the hepatic and the splenic arteries in the treatment of cirrhosis of the liver is questioned. The results obtained in patients operated upon do not warrant its continued usage.[29] Better results have been obtained by making a shunt between the portal vein and the inferior vena cava, but even this is still a last resort procedure.

ANTIBIOTICS

Antibiotics should not be looked upon as substitutes for the established forms of treatment, but rather as adjuncts. After a single intravenous injection of aureomycin, the drug is excreted by the normal liver in high concentration. The ability of the liver to excrete the drug is obviously impaired in the presence of obstructive jaundice or poor liver function.[30] Results with aureomycin to date seem to indicate that it should be given a therapeutic trial in patients with hepatic insufficiency.[31]

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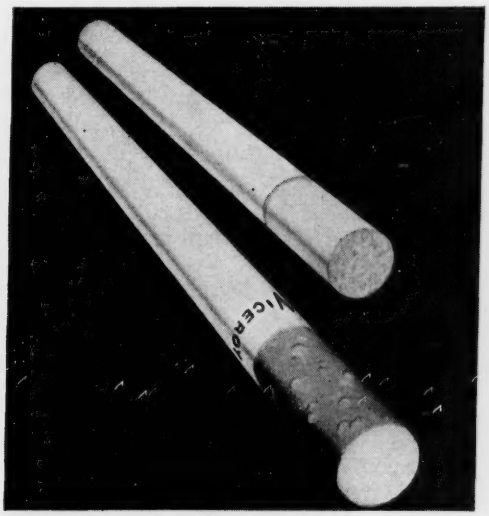

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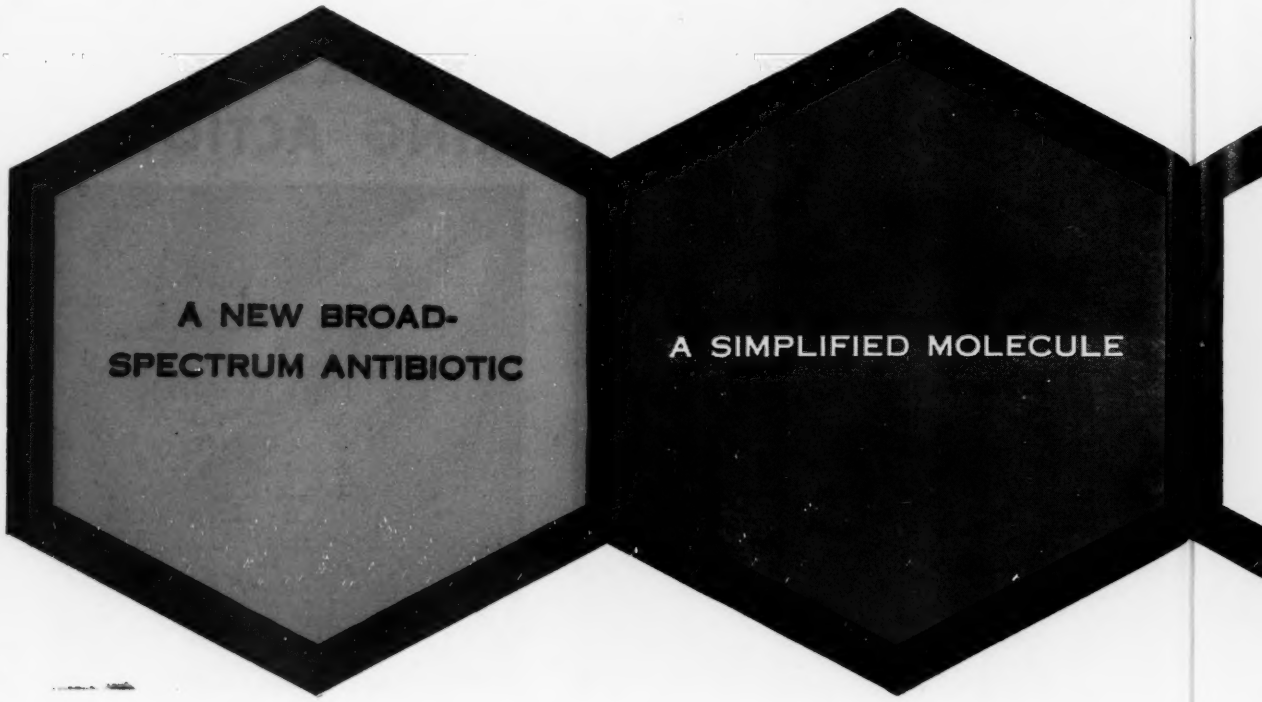

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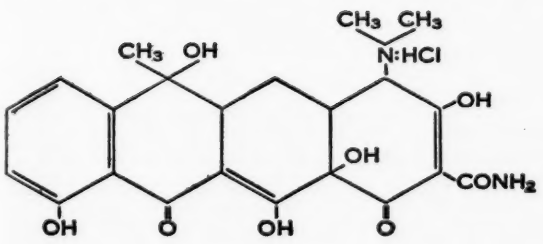

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striking to suggest that aureomycin may well have been responsible for the change, while in the fourth it was difficult to attribute the improvement to any therapy other than the parenteral aureomycin.[32]

If there is reason to suspect from the clinical course or laboratory findings that necrosis is a feature in a patient with coma, it would seem advantageous to administer aureomycin intravenously. Conversely, recent studies have suggested that large doses of aureomycin administered intravenously may actually cause fatty infiltration of the liver.[20] There is evidence that aureomycin administered intravenously to postoperative patients can produce transient clinical icterus, which can be differentiated from icterus from other causes by the observation of its prompt disappearance after the drug has been discontinued.[33]

Aureomycin, when added to a necrotizing yeast diet, has a significant beneficial effect on the prevention of experimental hepatic necrosis in rats. The assumption is made that aureomycin might act through the suppression of the intestinal flora. In the absence of sulfur-containing amino acids or of vitamin E as detoxifying agents, the products of uninhibited intestinal flora may be responsible for experimental acute dietary hepatic necrosis.[34]

ANEMIA

The anemia of liver disease is usually normocytic or macrocytic but may be microcytic when hemorrhage is present. The degree of anemia may be more apparent than real because of an increase in circulating plasma volume without an accompanying increase in total red cell mass. Wintrobe suggests that macrocytic anemia may be due to a variety of causes, such as inability to store hemopoietic principle, deficient diet, and faulty absorption, but that the defect is not related to pernicious anemia. Huang and Wang have suggested that destruction of blood in an enlarged spleen may be a factor. Reports are variable concerning response to vitamin B_{12} and liver extract.[20] Blood replacement therapy may be necessary in the case of extreme hemorrhage, e.g., esophageal varices.

SEDATIVES

By far the greatest danger to patients exhibiting psychotic and motor manifestations of hepatic failure is the indiscriminate use of sedatives. All of the commonly used sedatives, with the exception of bromides and possibly the slowly acting barbiturates, chloral, paraldehyde, and tribromethanol, have been found to exert an unpredictably prolonged and profound effect on patients with grave disease of the liver and commonly precipitate the terminal deep coma in patients who were showing only early evidence of cerebral dysfunction prior to sedation. It seems likely that these drugs in the doses commonly used do not exert any specifically noxious influence on the liver itself, but rather further depress metabolism of neurones gravely damaged by "poisons" or defects in nutrition and impairment of the inactivating functions of the liver.[16]

Analeptics, including glucose, sodium succinate, caffeine, and benzedrine, are without effect upon the comatose state.[35]

SUMMARY

It may be seen that the greatest problem in the treatment of hepatic insufficiency is that the organ affected possesses such reserves of function that it is not until damage is relatively advanced that clinical signs of impairment become apparent. By clarifying the natural history of different lesions, prognosis is placed on a firmer basis. By relating one pathological form to another, the initial lesion of each sequence can be recognized. It is by directing therapeutic and preventive measures against such early stages that the most effective results can be expected.[1]

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Epidemic Hemorrhagic Fever in Korea

The recent campaign in Korea brought to light an infectious disease never before noted among American troops by military physicians—epidemic hemorrhagic fever.

During the period 1932 to 1935 Russian doctors observed in certain parts of eastern Siberia occasional outbreaks of a disease which they termed hemorrhagic nephroso-nephritis or endemic hemorrhagic nephroso-nephritis. Japanese doctors recognized the disease during Japanese army operations in the Songo district in 1939, and later applied terminology which included typical scarlet fever, hemorrhagic purpura, typhus, acute nephritis, war nephritis, and fever disease.

The disease, actually, has never been a serious threat to American troops in Korea. The incidence rates have generally been low with the highest, approximately 18 cases per thousand average strength per year, coming in November 1951. Incidence has been spotty in military units in the field, and there has been no evidence that the disease can be transmitted from one person to another. A few more than 2,000 definitely diagnosed cases have occurred in American troops.

The disease is characterized by symptoms of moderately severe febrile illness, changes in capillary permeability, evidence of renal damage, hypotension, and hemorrhagic phenomena. Most cases follow a fairly typical course in the following phases: febrile, hypotensive, oliguric, diuretic, and convalescent. Treatment is largely supportive and is based on presenting symptoms. Antibiotics, sulfonamides, vitamins, hormones, and antihistaminics have little effect upon the course in epidemic hemorrhagic fever.

Since the first definitely diagnosed case was detected in Korea in 1951, the Army Medical Service has been engaged in a continuous research program designed to determine the causative agent of the disease, the most successful means of treatment, and a means of prevention.

New Chairman of Department of Surgery

Dr. Frank F. Allbritten, Jr., formerly associate professor of surgery at Jefferson Medical College, Philadelphia, became professor of surgery and chairman of the Department of Surgery at the University of Kansas School of Medicine on May 1. He succeeds Dr. Paul W. Schafer, who resigned to enter military service.

A native Kansan born in Cunningham, and an alumnus of the University of Kansas, Dr. Allbritten received his medical degree from the University of Pennsylvania in 1938. He spent two years there in internship, followed by three years as a resident at Pennsylvania Hospital. From 1943 to 1946, he served in the Army Medical Corps.

Dr. Allbritten has written or co-authored 23 scientific papers for medical journals reporting research and clinical results and two chapters for surgical textbooks. Much of his research has been with Dr. John H. Gibbon, Jr., on the artificial heart and surgical procedures to by-pass the heart while it is being repaired.

In addition to the usual medical society memberships, Dr. Allbritten is a diplomate of the American Board of Surgery, a fellow of the American College of Surgeons, and a member of the American Surgical Association, American Board of Thoracic Surgery, Society for Clinical Surgery, Society of University Surgeons, and the American Association for the Advancement of Science.

Dr. Charles M. Gruber, Jr., research associate with Eli Lilly and Company, Indianapolis, delivered the 18th annual Noble P. Sherwood lecture at the University of Kansas Medical Center, March 29. His subject was "Alcohol, Tobacco, and Medicine." The lectureship was established in honor of Dr. Noble P. Sherwood, now professor emeritus of the Department of Bacteriology at the medical school.

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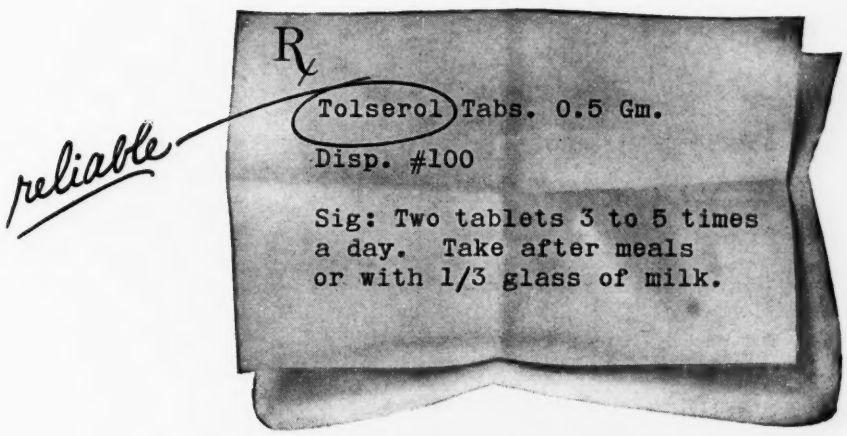

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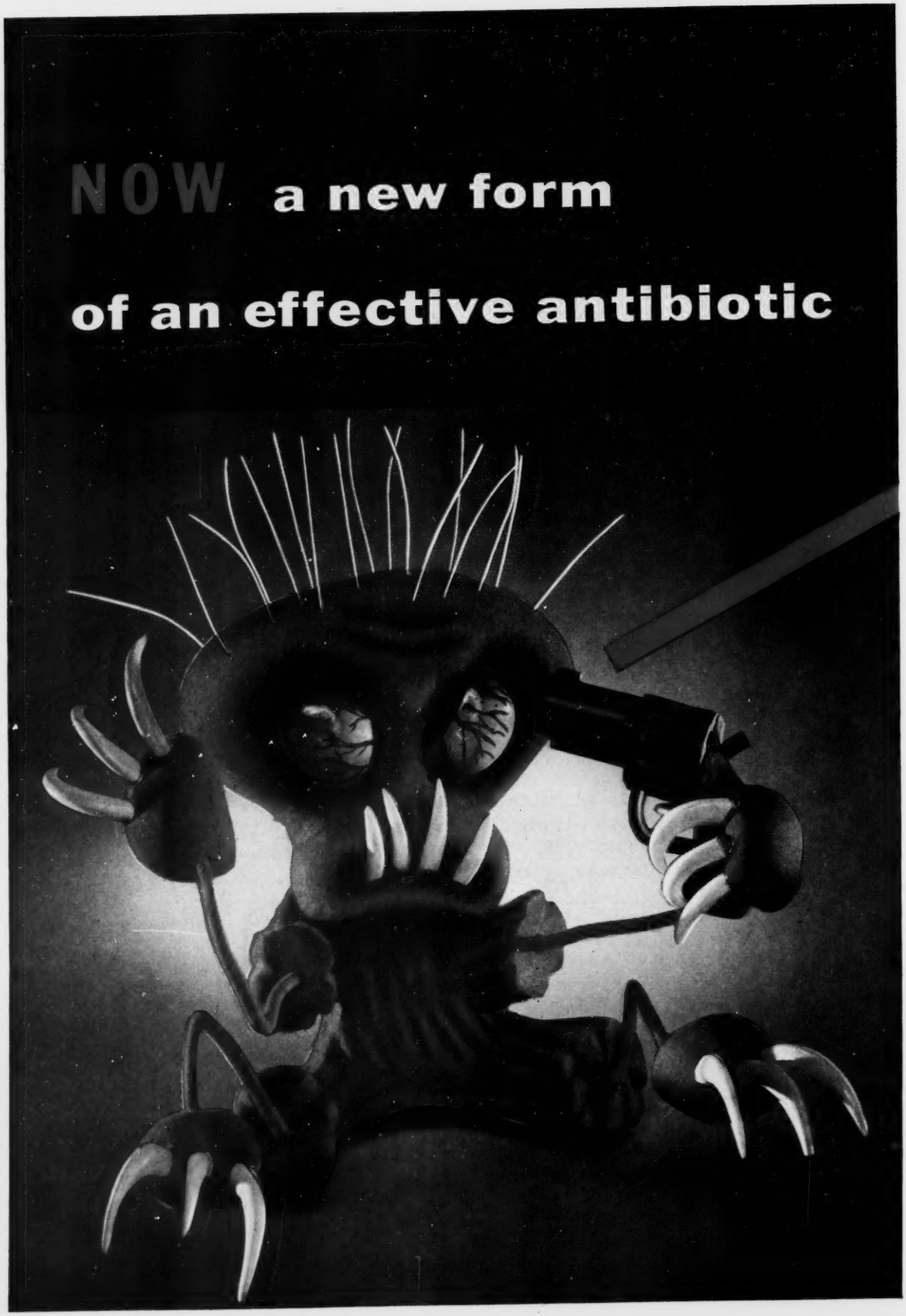
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BOOK REVIEWS

Children for the Childless. Edited by Morris Fishbein, M.D. Published by Doubleday and Company, Inc., New York. 223 pages. Price $2.95.

This book consists of a group of topics related to the problem of infertility. The authors are distinguished men in their field, and write with a great deal of authority and experience. The edition has been ably edited by Dr. Morris Fishbein, who also writes one of the chapters.

The book begins with discussion of the sociological problems of infertility and carries through the physiological, psychosomatic, and anatomical problems as related to infertility. There is a special chapter devoted to artificial insemination and its legal problems. The last two chapters review the problem of adoption and discuss some of the genetic factors associated with this problem.

This book can be a valuable adjunct to the physician who is seeing this type of problem. The book is also written so that the average layman could easily understand most of its contents. The chapters on psychosomatic aspects of infertility, adoption, and genetic characteristics of children are exceptionally good. They stress some of the most important problems that are frequently overlooked in this matter. The strictly sociological and scientific aspects of this book are at times confusing and mildly contradictory; only the superficial scientific aspects are covered, but this may be an excellent introduction for anyone interested in this problem.

One noteworthy chapter was written by Dr. Rubin, who was the originator of the Rubin test for tubal patency. His historic review of this subject is enlightening.—*R.H.O.*

The Hepatic Circulation and Portal Hypertension. By Charles G. Child, III. Published by W. B. Saunders Company, Philadelphia. 444 pages, 132 figures. Price $12.

This is a particularly appropriate book for those interested in disease of the liver. The text deals with embryology, anatomy, and physiology of the hepatic circulation, not touching upon the more general areas of physiology and metabolism in which the liver plays such an important part.

Chapter headings such as Comparative Anatomy of the Hepatic Circulation, Embryology of the Liver and its Blood Vessels, The Gross and Microscopic Anatomy of the Intrahepatic Vasculature, The Normal Physiology of the Hepatic Vasculature, The Liver and Shock, Portal Hypertension, Preoperative and Postoperative Water Electrolyte and Protein Balance in Patients with Cirrhosis represent the thoroughness of treatment by the author.

Commendable features of the book are its liberal usage of historical background material, recently accepted concepts of liver anatomy and structure, and excellent documentation of material by experimental evidence and a fine bibliography.

Every student interested in the liver, regardless of his field, will find this book useful.—*M.H.D.*

Salt and the Heart. By E. T. Yorke. Published by Edward T. Yorke, M.D., 1717 North Wood Avenue, Linden, New Jersey. 83 pages. Price $3.45.

The greatest value we find in reading this book is its presentation of an approach to the low sodium diet. The physician who finds it difficult to convince a patient of the value of sodium restriction might gain a lot from these pages. The author opens with a tale of an old sea captain who has developed a heart condition. It describes the various types of treatments used, stressing the low sodium diet and the captain's reaction to it.

The author also discusses briefly some of the symptoms of heart failure and hypertension, the history of the treatment of heart conditions, and the role of sodium in the body. Much of this discussion would be of value to the cardiac patient. However, one is inclined to wonder, while reading the book, exactly what type of reading audience the author had in mind. While much of it would seem to be addressed to the layman, there is considerable treatment of information on determining the levels of dietary sodium desirable in cardiovascular conditions. This type of information certainly has no business in the hands of the layman. Some of the dietary regimes outlined are inadequate in the nutrients recommended by the National Research Council, and one must question the value of urging malnutrition onto these patients. Aside from this, the book is written in an interesting style, and we should stress again its value in teaching the patient.—*M.A.P.*

Surgery of Infancy and Childhood, Its Principles and Techniques. By Robert E. Gross and William E. Ladd. Published by W. B. Saunders and Company, Philadelphia. 1000 pages, 1488 illustrations on 567 figures. Price $16.

This timely and up to the minute successor to *Ladd and Gross' Abdominal Surgery of Infancy and Childhood* is beyond doubt the best source of information concerning pediatric problems which necessitate surgery. The volume can be considered a reliable and complete reference to both the common

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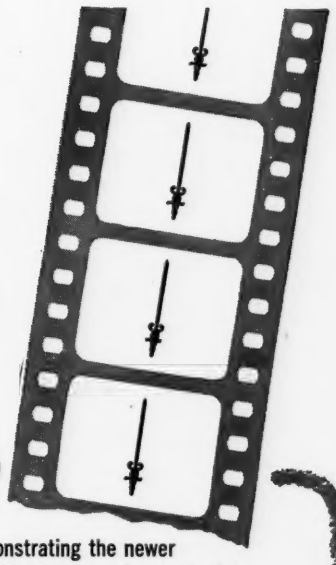

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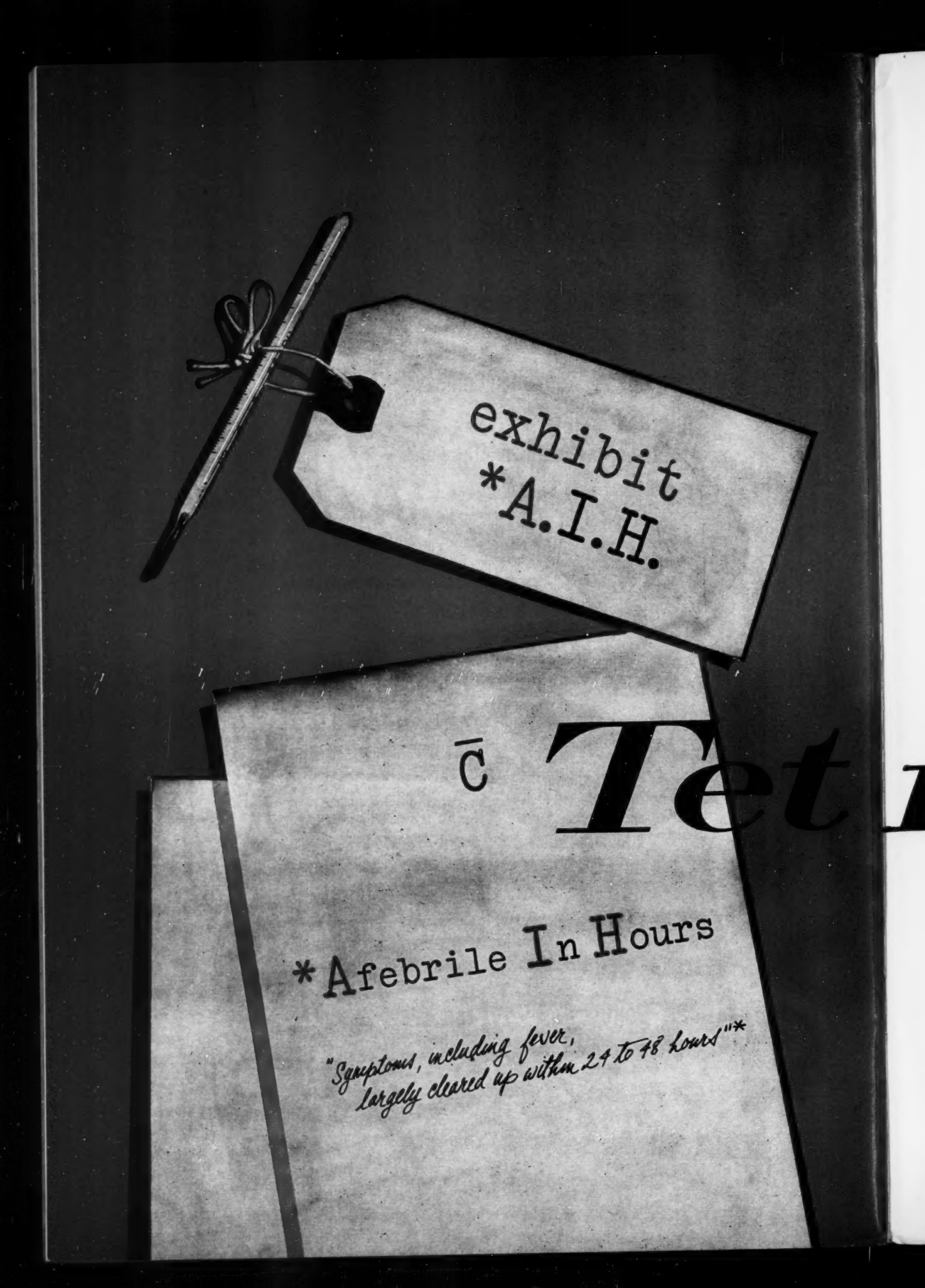
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and the unusual surgical conditions of infants and children. Any reader can sense the authenticity of the contents which reflect so much of the personal expeperience of the author, the undisputed leader in his field (2000 to 2500 general surgical operations are performed each year at the Boston Children's Hospital).

The large body of clinical matter is divided into 69 chapters; in most of these each chapter is concerned with a specific type of defect or disease, congenital or acquired. A few chapters deal more generally but pertinently with important aspects of pediatric surgery such as anesthesia for pediatric surgery, preoperative and postoperative care, surgery in premature infants, disruption of abdominal wounds, and rectal bleeding. The illustrations are excellent and clearly explained.

Although the author has not attempted to review the literature on each subject, he refers to pertinent work by others. He "describes for the most part the forms of therapy and management which [his staff] has come to feel are most suitable for the handling of the various diseases and abnormalities" of infants and children.

A complete report of the 95th annual meeting of the Kansas Medical Society, held in Topeka May 2-6, will be published in the June issue of the JOURNAL.

CONTRIBUTIONS FOR MEDICAL EDUCATION

A year-end report of the two fund-raising organizations for the nation's 79 medical schools—the American Medical Education Foundation, founded by the A.M.A., and its co-operating agency, the National Fund for Medical Education—was recently issued.

As of December 31, 1953 the American Medical Education Foundation, working through its 53 state and metropolitan committees, recorded a gross return of $1,089,962.93 from 18,176 individual physicians, organizations and laymen. This is the first time that the foundation's annual income has passed the million dollar mark. The number of contributors in 1953 increased 149 per cent over the previous year.

The National Fund for Medical Education, which seeks its funds primarily from business and industry, has just announced that during the past year 994 corporations contributed $1,367,979. This represents an increase over 1952 of 74 per cent in contributions and 193 per cent in the number of participating firms. This record is a tribute to the leadership of 1,000 corporation executives who make up the 47 industry divisions and 17 local sponsoring committees of the Committee of American Industry.

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Study for Physicians

The average practicing physician, in these busy times, still manages to devote the equivalent of 83.3 eight-hour days a year to keeping abreast of current developments in the field of medicine. This figure is one of many brought out in a preliminary report by the A.M.A.'s Council on Medical Education and Hospitals on its recent survey of postgraduate medical education.

Survey findings are based on data compiled on visits to more than 220 institutions engaged in postgraduate medical education as well as 5,000 questionnaires received from a random sample of 17,000 practicing physicians throughout the country.

Not only are more opportunities for postgraduate work being offered today than ever before, but more doctors are taking advantage of these opportunities. Over 41,000 physicians took some form of postgraduate course last year.

Other highlights of the preliminary report: Some form of organized postgraduate medical course is being offered in every state. . . ..Ninety per cent of these courses are offered in the larger cities. . . . More than 93 per cent of the responding physicians felt that the maximum time they could be away from practice was under 15 days. . . . It cost the average physician surveyed in this study approximately $350 per year to attend postgraduate courses. . . . Chief reason given by those not taking postgraduate courses is lack of someone to care for patients while the doctor is away.

The 103rd annual meeting of the American Medical Association will be held in San Francisco, June 21-25, 1954.

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ANNOUNCEMENTS

The 32nd annual scientific and clinical session of the American Congress of Physical Medicine and Rehabilitation will be held September 6-11, 1954, at the Hotel Statler, Washington, D.C. Sessions will be open to all physicians in good standing with the A.M.A. Information may be obtained from the Executive Office, 30 North Michigan Avenue, Chicago 2, Illinois.

The program of the first annual meeting of the Academy of Psychosomatic Medicine, to be held at the Plaza Hotel, New York City, October 8-9, will be devoted to psychosomatic aspects of surgery. Those interested in presenting papers should write Dr. Benjamin Raginsky, 376 Redfern Avenue, Montreal, Canada.

Applications for fellowships and associateships are now being received. New fellows will be inducted at the October meeting.

The sixth American Congress on Obstetrics and Gynecology will be held at the Palmer House, Chicago, December 13-17, under the sponsorship of the American Academy of Obstetrics and Gynecology. Information may be obtained from the Congress, 116 South Michigan Avenue, Chicago 3, Illinois.

Conference on Religion and Psychiatry

In an effort to improve relations between religion and psychiatry, and to encourage the interchange of ideas between representatives of the two groups, eight prominent theologians and nine psychiatrists will meet in Topeka, May 13 and 14. A semi-public meeting will be held the evening of May 13.

Bequest to Clendening Library

A bequest of $1,000 from the late Dr. Edward H. Skinner for the Clendening Library of the History of Medicine of the University of Kansas School of Medicine was announced last month by Dr. W. Clarke Wescoe, dean of the school. Dr. Skinner, who died January 11, 1953, had made other important gifts to the library, including 350 books, some of which are rare and of great value.

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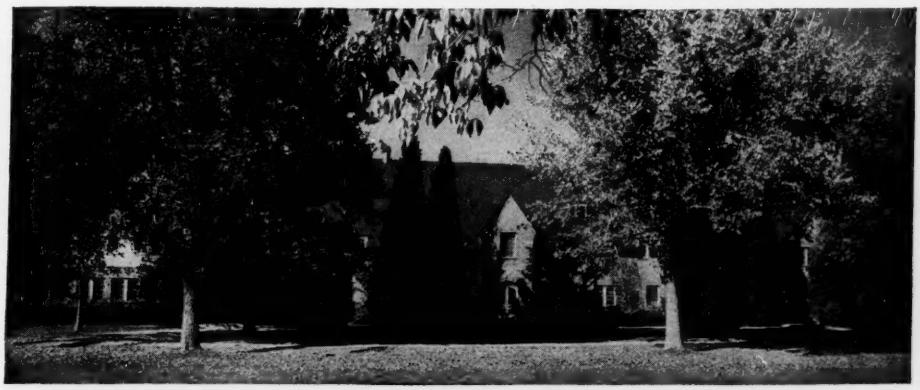

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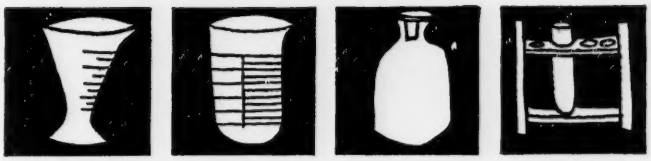

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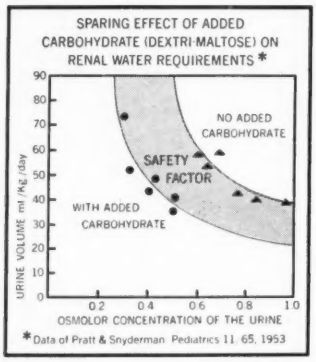

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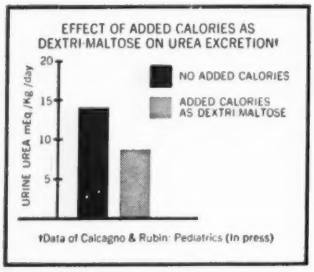

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